GROUP B STREPTOCOCCI IN PREMATURE RUPTURE OF MEMBRANES AND NEONATAL OUTCOME

Dissertation submitted

In partial fulfillment of the requirements for the degree of

M.D BRANCH II

OBSTETRICS AND GYNAECOLOGY

Tirunelveli Medical College

The Tamilnadu Dr. M.G.R. Medical University

Chennai, Tamilnadu

April 2011

CERTIFICATE

This is to certify that the dissertation entitled "**GROUP B STREPTOCOCCI IN PREMATURE RUPTURE OF MEMBRANES AND NEONATAL OUTCOME"** is the bonafide original work of **Dr. P.Pamela Packiavathi,** under the guidance of **Prof. Dr.Ramola Janet Diana. MD,DGO, HOD,** Department of Obstetrics and Gynecology**Tirunelveli Medical college** ,Tirunelveli in partial fulfillment of the requirements for the degree of M.D branch II Obstetrics and Gynecology examination of the Tamilnadu Dr. M.G.R Medical University to be held in April 2011.

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This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the requirement for the award of M.D. Degree, Branch II (OBSTETRICS & GYNAECOLOGY) degree Examination to be held in April 2011.

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ACKNOWLEDGEMENT

I am extremely thankful to **Prof. Dr. N.Palaniappan, M.D., Dean,** Tirunelveli Medical College and Hospital, Tirunelveli who has granted permission to do this study in this institution.

I take this oppurtunity to express my deepest sense of gratitude to **Prof. Dr. Ramola Janet Diana M.D.D.G.O,** Head of the Department of obstetrics and Gynecology, Tirunelveli Medical College, Tirunelveli for encouraging me and rendering timely suggestions and guiding me through out the course of this work. I will be forever indebted to her for her constant support.

I am extremely thankful to **Prof. Dr. Revwathy M.D.D.G.O.D.N.B** for her extensive support, advice and guidance in the analysis and successful completion of this study.

I sincerely thank my **Registrar Dr. Meena, M.D.D.G.O.D.N.B** I am extremely thankful to all the Assistant Professors of the department of obstetrics and gynecology for their guidance and support.

I wish to thank **Prof. Mr. P. Arumugam,** statistician for his useful inputs. I wish to express my gratitude to my friends and colleagues who have always been a source of love, support and encouragement. Above all my sincere thanks to antenatal mothers of Tirunelveli Medical College Hospital without whom this study would not have been possible.

ABBREVIATIONS

CDC	-	Centre for Disease Control
GA	-	Gestational Age
GBS	-	Group B Streptococcai
LSCS	-	Lower Segment Caesarian Section
PPROM	-	Preterm Premature Rupture Of Membranes
PROM	-	Premature Rupture Of Membranes
PTL	-	Pre Term Labour
Wt	-	Weight

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INTRODUCTION

Group B Streptococcus is usually a commensal bacterium that asymptomatically colonizes the vaginal and rectal areas of 10-30% of pregnant women. In these women Group B Streptococcus can cause preterm labour, Chorioamnionitis, post partum endometritis, post partum wound infection and sepsis. At birth approximately 50% of infants who are born to colonized mothers will also become colonized on their mucosal surface and the skin. The vast majority of colonized newborns remain free of symptoms, where as 1% will develop invasive disease.

The incidence of preterm delivery is between 5% to 10%. Despite advance in perinatal medicine, the social and emotional cost of perinatal mortality and morbitity associated with preterm births is immeasurable. Hence ideally preterm labour should be prevented.

A wide spectrum of causes and demographic factors have been implicated in the births of preterm infants. Many investigators suspect that infectious organisms play an important role in the occurrence of preterm births (Romero et al; 1980) Intrauterine infection accounts for 47% of total preterm labour.

Many studies have examined the association between maternal colonization with Group B Streptococci and pregnancy outcome. Group B Streptococcus can cause chorioamnionitis and urinary tract infection. The incidence of chorioamnionitis in general population is 0.5% to 1%.

In patients with premature rupture of membranes, the incidence increases to 3% to 31% (Gibbs et al; 1980)

Whether Group B Streptococci are also a cause of preterm premature rupture of membrane and preterm labour has been the subject of conflicting reports. Various studies have been conducted and the largest controlled study of this issue was conducted as a part of the vaginal infection and prematurity study group (Regan & co-workers; 1991, Feikin et al; 2001, G.C. Di Renzo- Italy; 2006 study and Nomura et al 2006).

Epidemiological studies in India have shown lower colonization and infection rates in general. In the study done in 50% pregnant Indian women, 12% where reported to have Group B Streptococcus isolated form throat mid vagina and 10% had positive vaginal cultures alone. Another study showed the over all carriage rate in pregnant women to be 16%. Colonization rates in infants born to asymptomatic maternal carriers of Group B Streptococcus is 53% to 56%. Despite significant Group B Streptococcus colonization rates, reports of invasive neonatal Group B Streptococcus disease in India are infrequent.

So the present case control study was planned clinically to evaluate the roll of Group B Streptococci in preterm labour and neonatal outcome.

HISTORY

As early as the 14th century sonarus and Hippocrates who were considered to be the great gynaecologists of the day made references about early and untimely rupture of membranes and its complication – dry labour.

Rosslin (1517 – 1526) wrote many articles in obstetrics. He attributed many a difficult labour to early rupture of membranes.

Abnormal Vaginal flora were considered an important cause of obstetric adverse sequelae as preterm premature rupture of membranes and preterm labour. The discovery of lactobacillus in baginal secretion by Albert Doderlien in 1892 marked the beginning of extensive research into the detailed composition of the vaginal flora.

However the emphatic demonstration by Semmelweis in 1847 that hospital outbreaks of puerperal fever could be prevented by the simple measure of hand washing by those attending the labour ward remains a landmark in clinical microbiology.

Since then various studies were carried out regarding relationship of abnormal colonization to preterm premature rupture of membranes according to gestational age at screening.

In 1875, a case was reported of delivery 90 days after rupture of membranes. Gordon and Pyle (1937) also referred to a case where there was premature rupture of membranes at 6 months gestation, but the

pregnancy was carried upto term subsequently resulting in a live birth. Baudeloque in 1789 held a gloomy prognosis for dry labour.

Broose (1961) remarked that there is a difference of opinion as to whether maternal morbidity is significantly altered in preterm rupture of membranes, but increased perinatal mortality is unquestioned.

Lebherz (1961) in a prospective study, concluded that perinatal mortality is significantly increased in cases with premature rupture of membranes. His results were in agreement with those of Eastman (1966).

Gunn, Mishell and Morton (1970) consider premature rupture of membranes as a threat to both the mother and baby.

The role of Group B streptococci in preterm premature rupture of membranes was studied by Regan et al. (1981) followed by Thomsan et al. (1987) Shoom Marker et al. (1989). GL Di Renzo et al.(1998 & 2006) Feikin et al. (2001) Tsolin et al (1998) and Nomura et al (2006).

REVIEW OF LITERATURE

Incidence

In industrialized world the incidence of preterm delivery rate lies between 5 -10% (Rush 1979 – Newzeland Health statistics report 1978). The incidence in India being 10 – 14%. The incidence of preterm premature rupture of membranes varies between 2 – 14% (Lebherz et al.1969; Akhthar et al, 1990) and is responsible for 30% of all preterm deliveries. The incidence of preterm premature rupture of membranes in the pregnant population is about 1% (Gibbs and Blanco 1982).

The role of infection as a cause of preterm labour has been a matter of conflict. But however growing evidence suggests that infection is associated with preterm premature rupture of membranes.

Colonization of genitourinary by group B streptococci is found to be associated with preterm premature rupture of membranes. The Vaginal Infection and prematurity Study found that 14.3% of women with group B streptococci colonization had preterm delivery as compared to 7.4% in women who delivered at term.

TABLE – I

Studies reporting incidence of premature rupture of membranes

Reference	Study interval	Incidence %	Study type
Flower et al	1954 – 56	15.80	Retrospective
Gupta et al	1956 – 66	10.73	Retrospective
Lebherz et al	Before 1960	07.08	Prospective
Lebherz et al	1960 – 61	11.54	Prospective
Bruzin et al	1980 – 81	09.83	Retrospective
Bourgeois et al	1980 – 85	07.35	Retrospective
Akhtar et al	1980	03.30	Prospective
Sanyal et al	1990	14.30	Prospective
GC Di Renzo et al	2006	5.4 – 6.5	Prospective

INFECTIONS AND PRETERM LABOUR

Infectious organisma play an important role in the occurrence of preterm birth. Microorganisms may gain access to the intra amniotic cavity by the following path ways :

1. Ascending infection from vagina and cervix
2. Haematogenous
3. Retrograde seeding form peritoneal cavity
4. By accidental introduction at the time of intrauterine procedures.

Bobitt and Ledger first suggested that unrecognized chroniamnionitis may be causally related to preterm labour. They documented positive cultures during amniotomy with transcervical needle aspiration or intrauterine catheter. Indirect evidence suggests that the most common pathway of intrauterine infection is the ascending infection.

Stage I

The first stage in the process of ascending infection is excessive growth of organisms in the vagina (vaginosis) and cervical canal.

Stage II

Once they gain access to the intrauterine cavity they reside in the deciduas leading to deciduitis and further extension to chorionitis.

Stage III

The organisms invade the fetal vessels or proceed through amnion into the amniotic cavity.

Stage IV

Once in the amniotic cavity the bacteria may invade the fetus by different ports of entry.

A localized infection in the chorio decidual junction may lead to rupture of membranes.

The effect of bacterial proteases, host products or both secreted in response to bacterial infection would lead to weakening of the membranes.

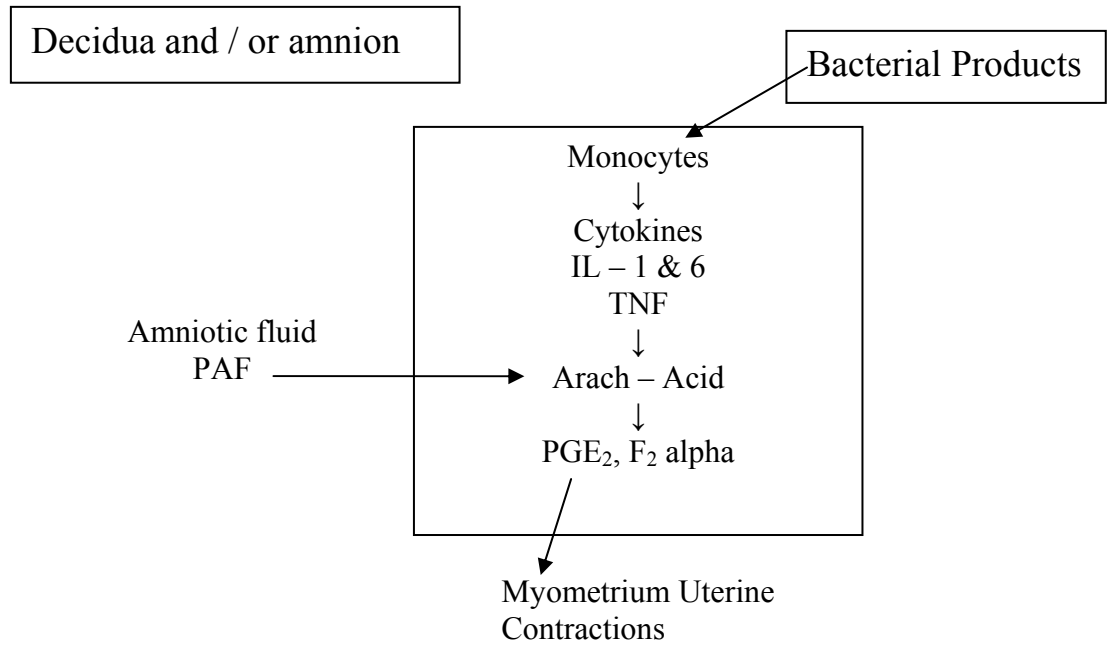
Biochemical techniques have shown that there is a reduction in the collagen content of prematurely ruptured amnion and the connective tissue layer contains a decreased number of poorly organized collagen fibrils. Specifically preterm premature rupture of membranes is associated with a reduction in type III collagen. Moreover the collagenolytic activity in premature rupture of membranes patients is found to be enhanced. Amniotic fluid contains trypsin which is proteolytic and also alpha-1-anti trypsin as its primary anti tryptic factor. In patients with preterm premature rupture of membranes amniotic fluid concentration of trypsin is found to be raised and that of alpha-1-anti trypsin to be reduced (Kanayama et al, 1985).

Biochemical mechanism in initiation of labour

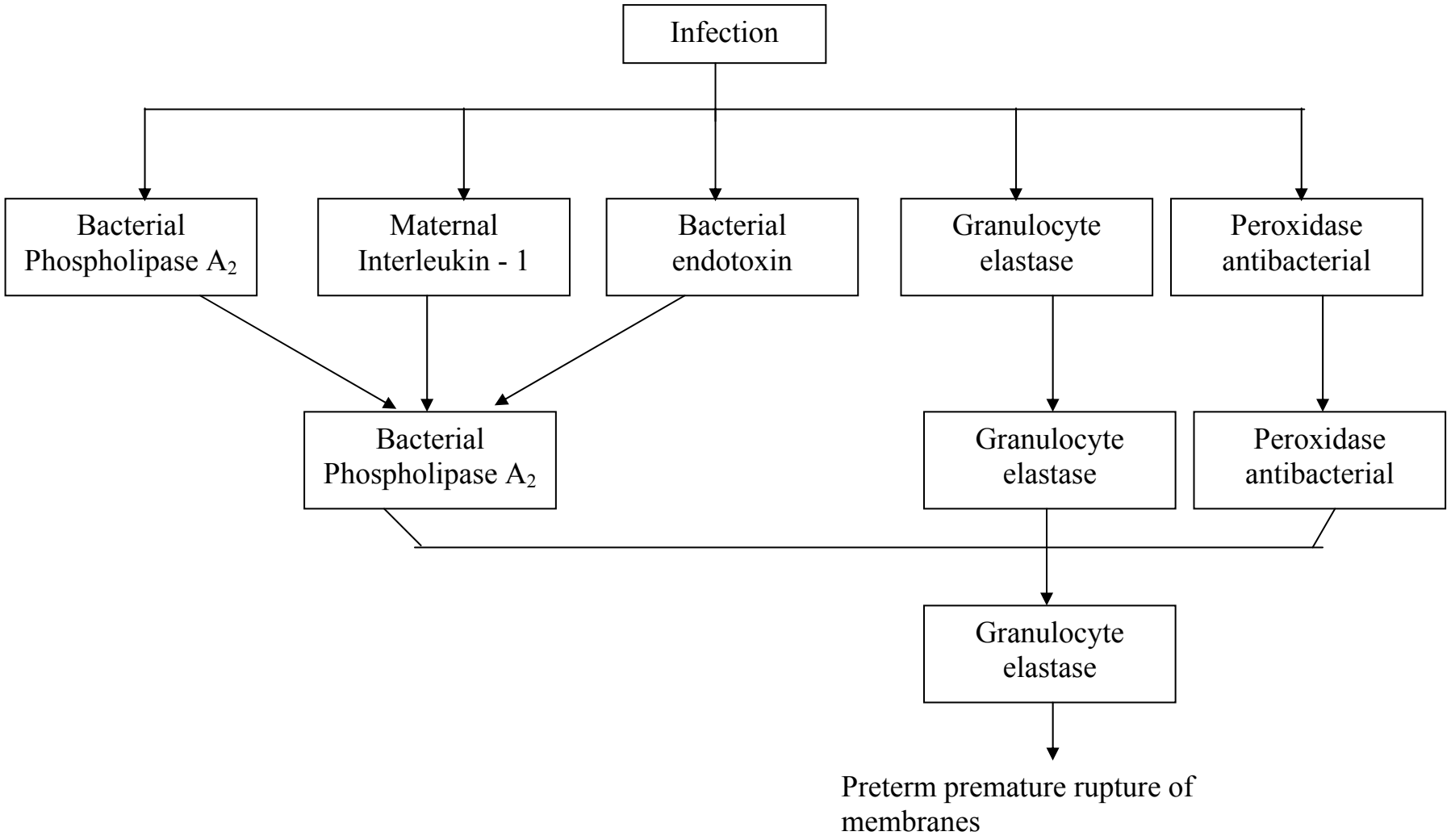
Schwartz and coworkers suggested that term labour is initiated by activation of phospholipase A_2. Which cleaves arachdoni acid from within fetal membranes for prostaglandin synthesis.

It has now been established that endogenous host products secreted in response to infection as Interleukin-1, Tumor Necrosis Factor and Interleukin-6 are implicated in preterm labour.

Proposed schematic mechanism of action for bacteria to incite preterm labour



MILLER AND PASTOREK PROPOSED MECHANISMS OF PROM – INFECTION MEDIATED



PROTEASE AND PROTEASE INHIBITORS ASSOCIATED WITH PREMATURE RUPTURE OF MEMBRANES

Human Proteases

Serine	Thiol	Carboxyl	Metallo
Trypsin	Cathepsin B	Pepsin	Collagenases
Chymotrypsin	Cathepsin N	Renin	Microvillus
Elastase	Cathepsin L	Cathepsin D	Protease
Cathepsin G			
Plasminogen activator			

Human Antiproteases

Alpha 1 antitrypsin

Alpha 2 macroglobulin

Alpha 1 antichymotrypsin

Collagenase inhibitor

Alpha 2 plasmin inhibitor

Intertrypsin inhibitor

Heat stable factor

Antithrombin III

RELATIONSHIP BETWEEN CERVICOVAGINAL COLONIZATION AND PRETERM LABOUR

Cervicovaginal system is dynamic and even the modest hormonal fluctuation of the menstrual cycle causes variations in the vaginal bacterial flora.

Normal bacterial flora

Aerobes	Anaerobes
E.Coli	Bacteroids
Lactobacillus	Clostridium
Gardnerella vaginalis	Peptococci
Staphylococcus epidermidis	Peptostreptococci
Staphylococcus aureus	Eubacterium
Streptococcus	

Increasing gestations bring down the number of anaerobic organisms, at the same time increasing the number of facultative organisms. The natural protective forces alter the vaginal flora in pregnancy to an extent that the fetus is well protected form more virulent organisms. The commonest organisms present during pregnancy in the vaginal flora is lactobacillus.

However colonization of genitourinary tract with several microorganisms has been associated with preterm labour and premature rupture of membranes. Though there seems to be a never ending list of

organisms labeled as probably responsible for premature rupture of membranes it is of importance to determine if a cause and effect relationship exists. The most important criteria to establish this relationship are study design and the differences in culture sites. Many studies have examined the association between maternal colonization with group B streptococci and pregnancy outcome. The present study reveals that women colonized with group B streptococci at delivery were 3 times more likely to have a preterm labour than were women who were not colonized.

Relationship of abnormal colonization to preterm delivery

Study	Year	Gestational age at screening	Relative risk
Gravett et al	1986	32	2
MacDonald et al	1992	28	1.8
Hiller et al	1995	26	1.4
Krohn et al	1995	26	1.5
Germaine et al	1994	26	1.2
Mc Gregor et al	1990	24	2.0
Ridvan et al	1993	20	2.0
Hay et al	1994	20	5.5
Regan et al	1996	20	5.6
Feikin et al	2001	20	3.0

The increased risk of premature rupture of membranes in patients with group B streptococci, Bacteroids and trichomonas vaginalis have been proved beyond doubts. The high incidence of pathogenic or potentially pathogenic cervical bacteria in pregnancy is related to chorioamnionitis and premature rupture of membranes. Bacteroides fragilis, peptococci and fusobacterium, bacteria commonly isolated from amniotic fluid in the presence of preterm labour and other common vaginal bacteria including lactobacilli and staphylococcus epidermidis have significant biochemical action which weaker the membrance (Creastas et al, 1981)

Evadson et al in 1982, demonstrated that in most women with premature rupture of membranes, the membranes as well as the placentae showed heavy bacterial invasion. Bacterial distribution within the membranes showed a choriodecidual preponderance. Ascending infection appeared to follow the choriodecidual route and may be the prime pathogenic event in many instances of premature rupture of membranes. The anaerobic bacteroides fragilis which is infrequently isolated in normal pregnant women was found is 5 out of 15 women with premature rupture of membranes.

Recent studies suggest that the increased intravaginal concentration of ureaplasma urealyticum and other microorganisms associated with bacterial vaginosis may be associated with premature rupture of

membranes, preterm labour and amniotic fluid infection may be a more important determinant of adverse pregnancy outcome than the simple qualitative recovery of the microorganisms from the maternal genital tract. Infection within the cervix and the lower uterine segment associated with vaginal infection may mediate some instances of premature rupture of membranes (Gravet et al, 1986).

Bobitt et al, and Lamone et al reported that colonization of the genital tract with group B streptococci is associated with preterm labour Regan et al & Feikin et al.

Nalye et al, in 1980 proved that amniotic fluid infections seem to be a cause of premature rupture of membranes, because infections were 2 -3 fold more common when fetal membranes ruptured before labour started than when they ruptured just after the on set of labour.

Martonas et al, reported in 1989 a significant increment in the prevalence of premature rupture of membranes for patients with group B streptococci colonized in the vagina and or the rectum versus non carrier patients. This is an evidence for deciduities, chorioamnionitis or intra amniotic infection as causative of atleast some cases of premature rupture of membranes. Clinically, inapparent infection is identified in approximately in one quarter of women with preterm premature rupture of membranes.

Group B streptococci was isolated from 16% of patients with preterm premature rupture of membranes versus 4% of the control population ($P<0.05$) (Alger et al, 1988).

In another study, C. trachomatis was isolated from 44% of patients with preterm premature rupture of membranes versus 15% in the control group ($P<0.001$). This association was independent of infection with group B streptococci or N. gonorrhaea.

Urinary tract infection

Colonisation

Colonisation of the urinary tract by group B streptococci has also been found to be associated with preterm labour. High prematurity has also been found to be associated with asymptomatic bacteriuria (Kass, Robertson et al, and Wren).

TABLE – II

Organisms cultured in amniotic fluid in women with premature rupture of membranes

Streptococcus	Agalactiae
Streptococcus	veridans
Staphylococcus	aureus
Hemophilius	influenzae
Baceteriodes	fragilis
Baceteriodes	melanonogenicus
Peptococcus	Sp.
Pepto streptococcus	Sp.
Furobacterium	Sp.
Gardenrella	vaginalis
Candida	Sp.

Group B Streptococci (GBS)

Group B Streptococci or Streptococci agalactiae are gram positive cocci which are beta haemolytic and catalase negative on blood agar plates. They are arranged in pairs or chains. Cocci is chains were first seen in wound infection by Billroth (1874) who called them streptococci (strepto, meaning twisted or coiled). Later Ogston in 11881 and Rosenback (1881) isolated these cocci. Brown (1919) categorized these streptococci into 3 varieties based on their growth in 5% horse blood agar pour plate cultures.

1. Alpha - Patial hemolysis
2. Beta - Hemolysis
3. Gama - No hemolysis

The hemolytic group were than classified into 20 Lancefield groups as A, B, C based on the nature of specific carbohydrate antigen on the cell wall. Group B Streptococci are further classified into 5 serotypes. Ia, Ib, Ic, II and III. The distribution of serotypes of group B streptococci isolated from vaginal culture reveals approximately one third of Ia, b or c, one third of II and one third of III. Sero type III accounts for about a third of all early onset Neonatal sepsis. Sero type II accounts for about quarter of all cases.

Group B Streptococci are important pathogens in cattle, producing bovine mastitis. From 1960's, Group B Streptococci has assumed great clinical importance as the single most common cause of neonatal meningitis in the West. Infection is acquired from maternal vagina during birth.

On an average 10% of all women will reveal group B streptococci colonization of genitourinary tract at the time of delivery. However only a few will have intrapartum or postpartum infections caused by group B streptococci. The incidence of colonization varies among different populations.

Gram stain was used for predicting the presence of group B streptococci in the patients with preterm premature rupture of membranes on staining of vaginal fluid and specificity was only 66%. Selective vaginal gram stain provides an effective and rapid screening method for identifying the presence of group B streptococci and allows for the immediate institution of appropriate antibiotic therapy in the event of on set of labour before the availability of culture results (Field et al, 1987).

The rapid diagnosis of intra amniotic infection in patients with premature rupture of membranes and preterm labour is of utmost importance. However smears are of no value in infections of genitalia where streptococci may from part of vaginal flora. The Gram's stain examination of amniotic fluid can detect only half of the infections. So, it

is suggested that a controlled therapeutic trial of action intervention in those cases of premature rupture of membranes with elevated C-reactive protein in the absence of other clinical parameters suggestive of intrauterine infection should be undertaken. (Korman et al, 1988).

On an average 10 – 30% of woman of childbearing age will reveal group B streptococci colonization of genitourinary tract. The incidence is higher when selective broth is used as culture media and when samples are taken from lower one third of vagina and rectum. The rectal sample is of particular importance because colonization of birth canal is secondary to colonization of anorectal region which is the major of group B streptococci by 25% over that with vaginal cultures alone. Sheep blood agar is recommended for primary isolation because it is inhibitory for Haemophilus, colonies of which may be confused with those of hemolytic streptococci.

Data from streptococci reference laboratories in India (Lady Hardinge Medical College, New Delhi; Christian Medical College, Vellore) showed that approximately 15% of hemolytic streptococci isolates tested belong to group B.

TABLE – III

Studies showing association between group B streptococci and preterm premature rupture of membranes

Reference	Study period
Regan et al	1981
Sbarra et al	1987
Thomsan et al	1987
Schoonmake et al	1989
Di Renzo et al	1998 & 2006
Feikin et al	2001
Nomura et al	2006

Maternal Risks due to group B streptococci

Unless the mother is grossly neglected, maternal mortality should not occur due to preterm premature rupture of membranes and group B streptococci. Even maternal morbidity is almost negligible. However development of chorioamnionitis or overt infection of the amniotic cavity is associated with high maternal morbidity and sometimes maternal death.

The sequelae of chorioamnionitis are pelvic inflammatory disease, peritonitis, sepsis and disseminated intravascular coagulation.

Postpartum infection

The mode of delivery influences the risk of postpartum infection. Caesarean section carries a higher rate of infection compared to vaginal delivery.

Failed induction in the presence of a poor cervical score will result in an increased rate of operative delivery.

The use of steroids, tocolytics and antibiotics may produce some side effects in the mother although the risk in minimal.

Psychological disturbances due to prolonged hospitalization or fear of poor outcome should never be underestimated.

Evaldson et al (1980) found that patients delivered 24 hours after premature rupture of membranes had significantly more puerperal infections than those with a latent period of 24 hours. The risk of antepartum and / or postpartum febrile morbidity increases. Maternal fever was found to be an unreliable prognostic indicator. The incidence of puerperal infection amounted to 27% in the premature rupture of membranes group.

Microbial invasion of the amniotic cavity occurs in approximately one third of patients with preterm premature rupture of membranes. With the rupture of membranes the clock of infection starts to tick Microbial invasion of the amniotic cavity is a risk factor for endometritis in women with premature rapture of membranes (Romers et al, 1992). In the

presence of infection, abnormal labour patterns are common and the chances of operative delivery are increased. especially in patients with an unripe cervix.

In rare cases of extensive infection, with uterine necrosis a life saving hysterectomy may be necessary. Occasionally, uncontrolled infection may lead to septicemia, shock, DIC, adult respiratory distress syndrome and maternal death.

Fetal / neonatal risks

The occurrence of preterm premature rupture of membranes during a pregnancy presents the paediatrician with a number of potential problems in the future management of the newborn. The consequences of preterm premature rupture of membranes for the neonate fall into three major overlapping categories of which the paediatrician needs to be aware:

1. Neonatal mortality and morbidity associated with prematurity.
2. Complications during labour and delivery that increase the potential for neonatal resuscitation and
3. Infection.

Infection in the newborn in acquired from maternal vagina during birth. Between 40 – 50% of infants born to women with positive intrapartum culture for group B streptococci will be colonized at the time

of delivery and exhibit positive surface cultures for the same maternal group B streptococci serotype.

Prematurity is a significant risk factor and preterm premature rupture of membranes accounts for 30 – 40% of preterm deliveries.

Sepsis is another important factor which plays a major role in neonatal morbidity and mortality.

Oligohydramnios over a long period (>4 weeks) in the previable period may result in postural deformities and lung hypoplasia. It may also cuase cord compression and fetal death or intraprtum fetal distress when the patient gets established in labour.

Abruptio placentae and cord prolapse may compromise the fetus and necessitate urgent operative delivery.

In a prospective study of group B streptococci carriage and disease conducted over 6 years the following observations were made. Preterm, prolonged labour, premature rupture of membranes more than 12 hours and maternal infection enhanced the risk of "early onset" group B streptococci disease. *"Early onset"* disease shows signs of sepsis as respiratory distress, apnoea and shock within 6 – 12 hours of birth. *"Late onset"* disease manifests as meningitis a week or more after birth. These cases are most often caused by serotype III organisms. The mortality rate is less for late – onset meningitis than for early onset sepsis. A maternal

source of infection was identified in 34 of the 45 infants (Dillon et al, 1987).

The attack rate is only a fraction of the colonization rate and is directly related to the serverity of colonization. The incidence of early onset infections is 4/1000 in lightly colonized patients and 50/1000 when colonization is heavy.

The overall early onset infection rate in colonized newborns in <1%.

Madan et al. (1988) in a study of organisms isolated in preiprenatal autopsies due to chorio amnionitis found that the lung was the most frequent site cultured and the four most frequently isolated organisms were : Staphylococcus epidemidis 18% group B streptococci 13% E.coli 9% ureaplasma urealyticom 9%. Negative cultures from multiple sites occurred in 7% of cases (159 neonatal autopsies). This shows that multiorgan cultures help in defining the role of particular bacteria as pathogen.

An alalysis of all early onset neonatal group B streptococci infections for 10 years period, proved one or more predisposing perinatal risk factors evident in 82% of cases – premature labour 79% prolonged membrane rupture (> 12 hours, 57% premature rupture of membranes 69%, maternal sepsis 29%). Overall 88% of group B streptococci

infections were evident within 24 hours of birth, suggesting an intrapartum pathogenesis for infection (Gerland et al, 1991)

SCREENING PROGRAMMES

First strategy : The one that is recommended by centres for disease control and prevention . This involves taking swabs from rectum and vagina between 35 – 37 weeks, from all pregnant woman and offering intraparturm prophylaxis if they are identified as Group B Streptococci carriers. If women present in labour before swab has been taken or before the results are available, then intraparturm antibiotic prophylaxis is offered.

Second strategy : is risk based approach

In this women are not swabbed antenatally but are identified as being at increased risk of having a baby, who develops early onset Group B Streptococci if they present with risk factor such as preterm labour, prolonged rupture of membranes or have fever during labour.

Third Strategy : is a combination of the previous two.

In this approach, woman were swabbed antenatally and if they subsequently present with a 'risk factor' in labour such as prolonged ruptured membranes or fever, they are offered intra partum antibiotic prophylaxis.

Fourth strategy : is one of rapid bed side testing in labour. Women identified as being carriers are thus offered intrapartum antibiotic prophylaxis.

ROLE OF ANTIBIOTICS

CDC 2002 and ACOG recommendation

Patients with PPROM should have genital tract cultures obtained for group B streptococci. Penicillin G should be started after cultures are obtained with a loading dose of 5 x 10^6 units intravenously followed by a maintenance dose of 2.5 x 10^6 units every fourth hourly. If Penicillin G is not available 2gm loading dose of intravenous ampicillin should be started followed by 1gm every four hours. The purpose of this management is to decrease the vertical transmission of group B streptococci and the severe neonatal morbidity that may occur.

Clindamycin 900mg i.v. every 8 hours until delivery or erythromycin 500mg i.v. every 6 hours until delivery to those allergic to penicillin.

A recent study done at university hospital, Newark, New Jersey showed that after 3 days intravenous Penicillin G therapy group B streptococci was eradicated from the genital tract in women with PPROM from the time of admission to the end of the latency period. A total of 220 patients were included in the study. 46 tested positive for group B streptococci as admission. For all 46 patients, genital cultures were negative for group B streptococci by day 3. Therefore it was proposed that 3 days of Antimicrobial prophylaxis for group B streptococci should be given.

Cefazoline 2gm i.v. initial dose, then 1gm every 8 hrs until delivery or Vancomycin 1gm i.v. every 12 hours until delivery.

According to Cochrane Review, antibiotic prophylaxis results is a relative risk decrease of 83% of babies with confirmed early onset Neonatal infection.

The largest randomized control studies are the NIH maternal foetal Medicine collaborative group and ORACLE 1 randomized trial. In both studies the incidence of severe RDS, severe intraventricular Haemorrhage, Neonatal sepsis, Pneumonia and Necrotizing enterocolitis were reduced with the use of ampicillin or erythromycin.

Broad spectrum antibiotics, such as ampicillin should be avoided if possible as concerns have been raised regarding increased rates of Neonatal gram negative sepsis.

Topical Therapy

Chlorhexidine gel (0.2%) – vaginal flushing – one flushing every six hours until delivery – is efficacious in more than 90% cases at any stage of pregnancy.

VACCINES

The vaccination of women of child bearing age and the provision of protection by placental transfer of specific antibody appears to be an ideal solution for the prevention of Neonatal Group B Streptococci sepsis. The complexity of achieving this has been highlighted by the recent publication of the entire genome of Streptococcus agalactiae. This Group B Streptococci genome contains 2082 genes and the functions of 40% remain unknown.

The majority of vaccine strategies have involved conjugating capsular polysaccharides with T-Cell dependant protein antigens, such as Tetanus Toxoid or diphtheria toxoid, which enhance immunogenicity. Other groups have used novel conjugates such as C_{5a} peptidase, which is present in most Group B Streptococci isolates. Antibody to this peptidase provides, serotype independant killing of Group B Streptococci.

In a recent study, the risk of disease, correlated with the amount of protective normal maternal antibody present. When maternal antibodies levels were >= 5 /ml, there was an 88% risk reduction in invasive disease. Therefore it appears that not only does any vaccination have to maintain a certain level of antibody but this also has to be maintained for potentially many years, when that woman could bear children.

Vaccine trails in pregnant women would also be ethically difficult and large sample sizes would be needed to show an effect on neonatal sepsis rates as Group B Streptococci disease is still a relatively rare event.

Vaccination could also lead to reduction in Maternal Group B Streptococci urinary tract infection and chorioamnionitis and could potentially prevent preterm births.

AIMS

1. To study the association of group B streptococci colonization with preterm premature rupture of membranes in a tertiary care institute.

2. To evaluate pregnancy outcome in group B streptococci positive cases.

MATERIALS AND METHODS

This prospective case control study was carried out in the Department of Obstetrics and Gynaecology at Tirunelveli Medical College Hospital, Tirunelveli, during the period of 1 year from October 2009 to September 2010. 104 cases of preterm premature rupture of membranes were clinically evaluated and followed up. 96 cases delivering at term were taken as controls. Also the pregnancy outcome in group B streptococci positive cases was anlysed.

Inclusion creiteria

- The study group comprised 400 consecutive pregnant women with preterm premature rupture of membranes and delivering before 37 weeks of gestation attending the out patient department and then admitted in labour ward were included.
- Booked, unbooked and referral cases were included in the study.
- Both primi and multi were included.

Exclusion criteria

Women were excluded from analysis if they had

- Multiple pregnancy
- Placenta praevia
- Cervical incompetence treated with cervical encirclage
- Hydramnios

- Pregnancy induced hypertension

Clinical study

A thorough clinical evaluation was done before submitting them for investigation. A detailed history was taken covering the following aspects in each case. General particulars as name, age, socioeconomic status, marital status and date of admission were noted. An elaborate obstetrical history was taken with reference to the parity, period of gestation and previous history of premature rupture of membranes and preterm labour. In the previous history of premature rupture of membranes the ultimate maternal and fetal prognosis was carefully analysed. In the current pregnancy a detailed history of complications associated with pregnancy was taken. The time of occurrence of premature rupture of membranes was carefully noted. The cases were then clinically examined and the gestational age confirmed.

This was followed by a speculum examination which was done to detect preterm premature rupture of membranes. Samples were collected which include samples from the vaginal pool and anorectal swabs. These to specimens were sent for culture. Per vaginal examination to assess the sate of membranes and cervical dilatation. Antibiotics were started at admission itself for all cases of premature rupture of membranes. This was followed by elaborate investigations.

Routine investigations

1. Blood grouping and Rh typing
2. Haemoglobin estimation
3. Total leukocyte court
4. Differential white cell counts
5. Urine routine and microscopic examination
6. Specific investigations
    a. Vaginal pool culture
    b. Anorectal swab culture

After delivery

Skin swab culture and

Ear swab culture were collected for baby.

Laboratory technique

Samples were collected from the posterior fornix with a sterile nonlubricated vaginal speculum and samples were also collected from anorectal region by a sterile, cotton tipped wooden and stick. Samples were than transported to laboratory in Todd-Hewitt broth. Material from transport medium was then subcultured on specific blood agar plates and read for hemolysis.

Detailed records as per proforma noted. The Apgar score of new born at 1, 5 and 10 minutes were recorded. The baby was examined after delivery and the following were taken note of:

1. Weight of the baby
2. Degree of asphyxia
3. Apgar score
4. Maturity

Both the patients in study and control groups with their neonate were followed up, till discharge from the hospital. In the follow up of the babies, any neonatal morbidity in the form of infection, respiratory distress and convulsions were looked for. All cases of perinatal mortality were studied in detail with careful analysis to determine what factors were more consistently associated with fetal and neonatal death. At the same time, mothers were examined to detect any evidence of infection or any other morbidity.

The variables were studied in each group separately and the outcome was compared between group B streptococci positive and negative cases. The associate between discrete variables were evaluated by statistical analysis.

OBSERVATIONS

The study was conducted during the period of one year from October 2009 to September 2010 at TVMCH, Tirunelveli. During the study period, 104 cases of premature rupture of membranes and 96 cases of control groups were studied.

The association between group B Streptococci colonization and preterm rupture of membranes was analysed. Also the effect of group B Streptococci positivity in maternal and fetal outcome was analysed in the study group.

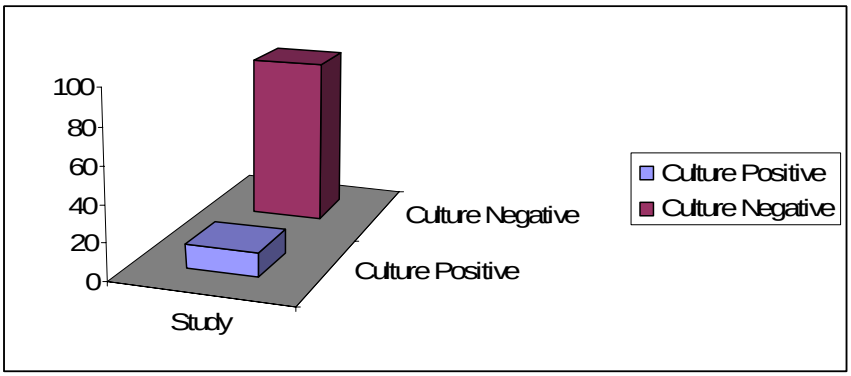
Out of 104 cases group B Streptococci was positive in 13 cases (12.5%) compared to 4 cases (4.2%) of culture positivity in 96 control group.

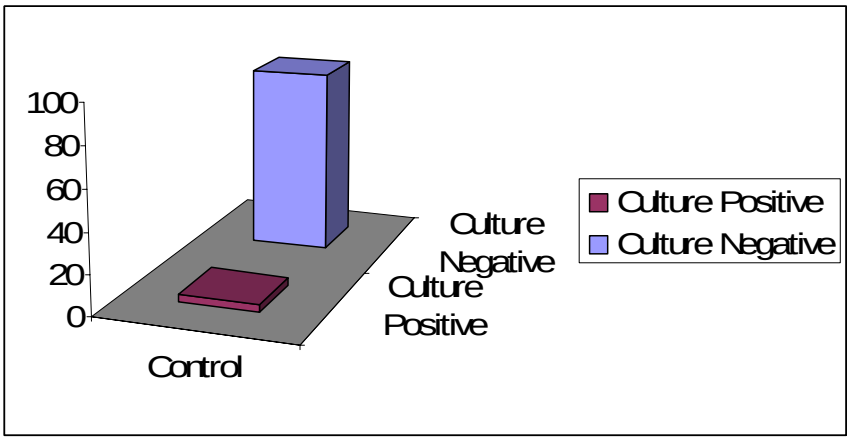
This shows that group B Streptococci were isolated significantly in patients with PROM as compared with controls.

Table – 1

Group B Streptococci in patient into PROM and controls

Group	Total	Culture Positive		Culture Negative		ψ^2	d.f	Significance
		No	%	No	%			
Study	104	13	12.5%	91	87.5%	4.45%	1	P<0.05
Control	96	4	4.2%	92	95.8%			



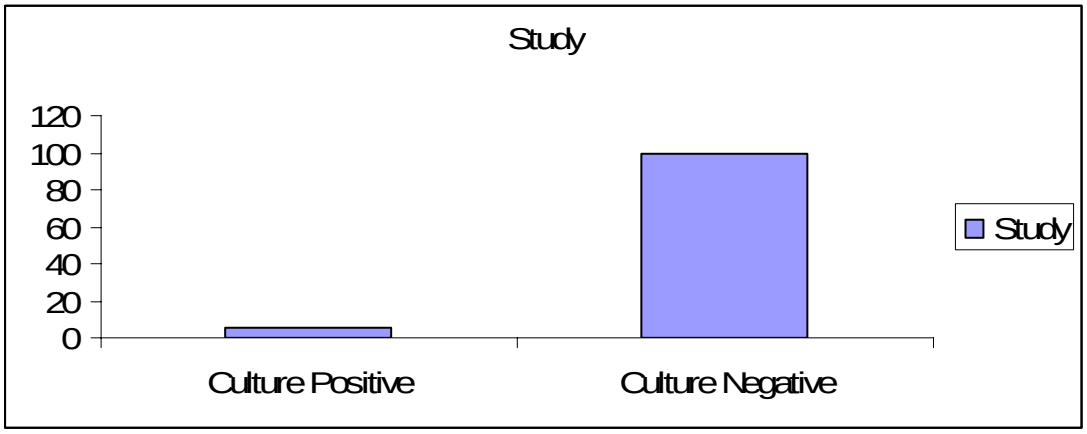


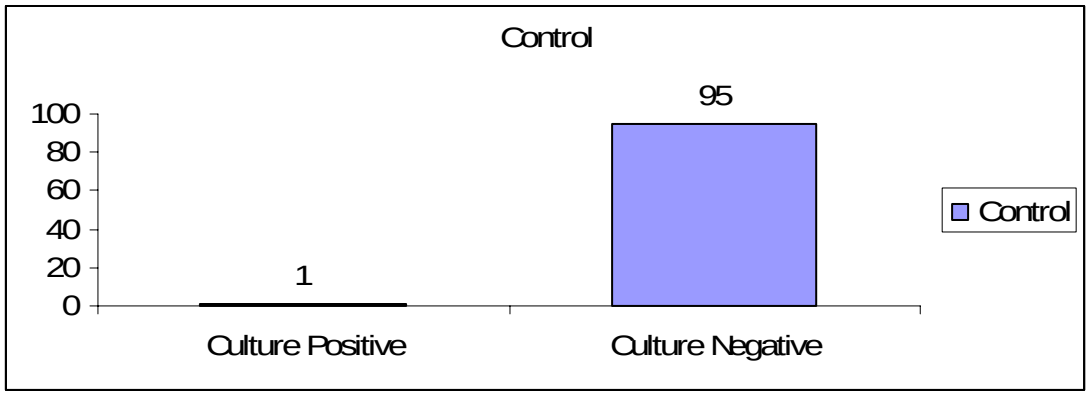
In the present study, out of 104 causes 13 cases ie 12.5% are Group B Streptococci positive. In control group only 4 cases ie 4.2% are Group B Streptococci positive. This is statistically highly significant.

Table II

Group B Streptococci in Babies with PROM and controls

Group	Total	Culture Positive		Culture Negative		Z	Significance
		No	%	No	%		
Study	104	5	4.8%	99	95.2%	1.631	P> 0.05
Control	96	1	2%	95	99%		





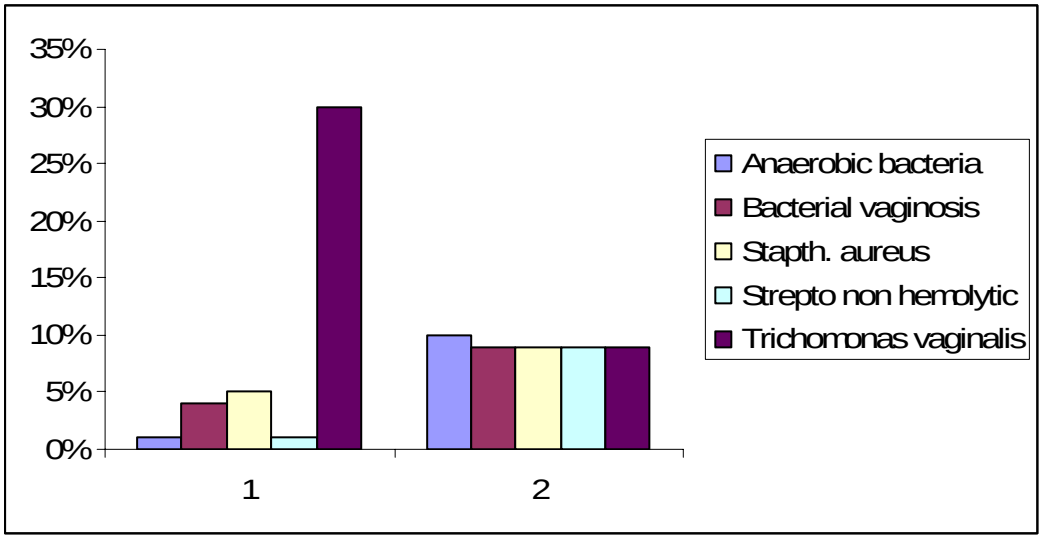
Out of 104 babies, Group B Streptococci was positive in 5 babies (4.8%) in study group as compared to 1 baby 1% in the control group.

But no infant in either group had culture proved bacterial sepsis in the first week of life.

Table – III

Relationship between vaginal colonization with GBS and other microorganisms

Microorganisms	GBS when other organisms (+)	GBS when other organisms (-)
	%	%
Anaerobic bacteria	1%	10%
Bacterial vaginosis	4%	9%
Stapth. aureus	5%	9%
Strepto non hemolytic	1%	9%
Trichomonas vaginalis	30%	9%



Our study supports the view that GBS is an opportunistic colonizer and more likely to be present in the absence of normal vaginal flora. It has been found that there is a high incidence of group B streptococci positivity when Trichomonas virginals were present.

Table IV

Multivariate Analysis of association between GBS and preterm delivery in case control study

Factor	Case		Control	
	No	%	No	%
GBS	13	12.5%	4	4.2%
High Risk behaviour	13	12.5%	4	4.2%
Previous Preterm Delivery	56	53%	10	10.3%
No higher education	12	11%	4	4.2%
IUCD	20	1.9%	3	3.1%
Strenuous Physical work	36	3.4%	20	2.0%

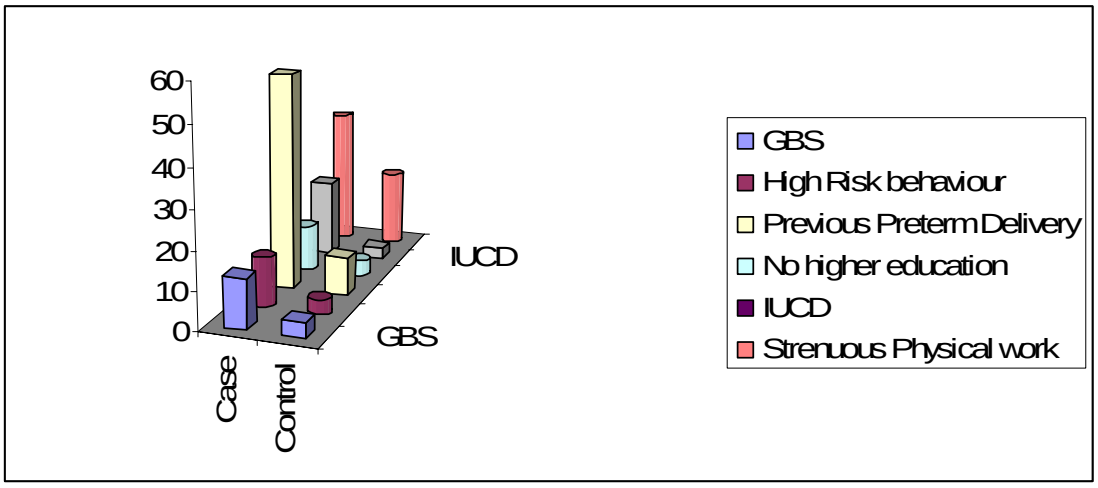
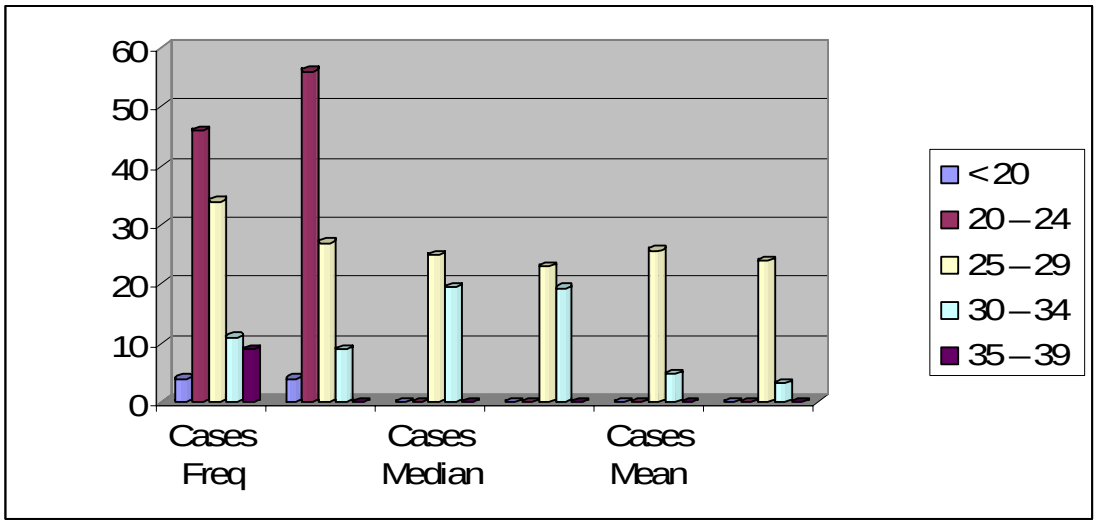


Table IV shows that previous history of preterm delivery was associated with PROM as noted by high incidence of 53% as against 10.3% in control group.

Table – V

GBS in patients with PROM categorized by Age

Age Group	Cases		Controls		Median		Median ± SD		t	ds	Sig.
	Freq	%	Freq	%	Cases	Controls	Cases	Controls			
< 20	4	3.8%	4	4.2%	-	-	-	-	2.996	198	p<0.05
20 – 24	46	44.2%	56	58.3%	-	-	-	-			
25 – 29	34	32.7%	27	28%	25	23	25.7	23.9			
30 – 34	11	10.6%	9	9.4%	19.39	19.32	4.9	3.2			
35 – 39	9	8.7%	-	-	-	-	-	-			
Total	104	100%	96	100%	-	-	-	-			



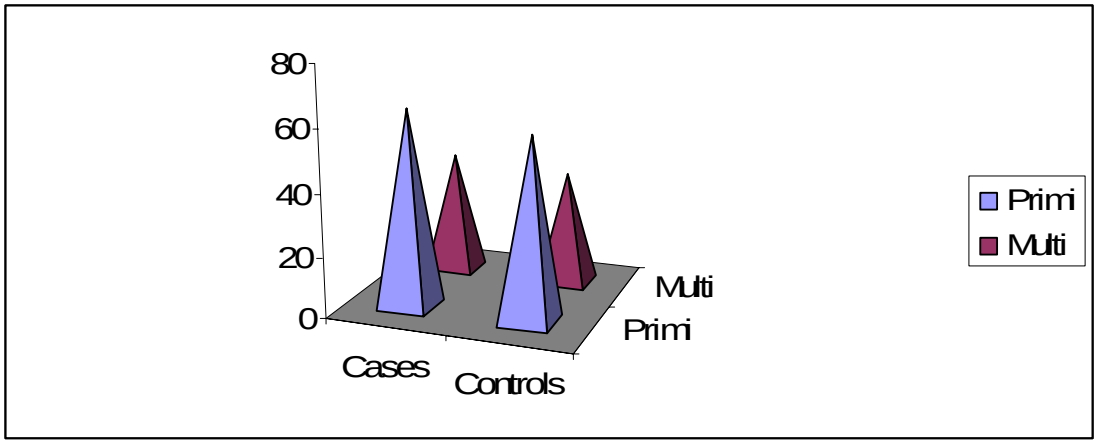
The mean age of culture positive patient with study group was 25.7 ± 4.9 compared to 23.9 ± 3.2 years in control group. This shows the cases were significantly older than the controls in this study.

Table VI

Shows the parity distribution in the study group

Distribution of cases and controls according to their parity

Parity	Cases		Controls	
	No	%	No	%
Primi	64	61.5%	59	61.5%
Multi	40	38.5%	37	38.5%
Total	104	100%	96	100%



The above table shows that the percentages of parity among the 2 groups were equal. So the 2 groups were matched and comparable in respect of their parity.

Table VII

Ante natal care

AN Case	Cases		Control		Z	Significant
	No	%	No	%		
Booked	84	80.8%	86	89.6%	1.773	
Unbooked	20	19.2%	10	10.4%		P>0.05
Immunization	104	100%	96	100%		

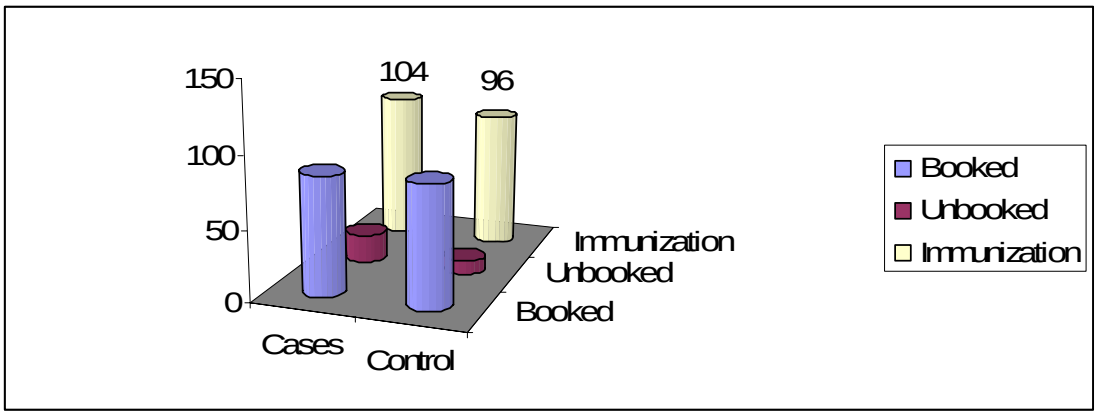


Table VII shows the level of booking of the study subsets. The booked and unbooked cases in the study group are 80.8% and 19.2% respectively.

The booked and unbooked cases in the control group were 89.6% and 10.4% respectively. By matching for factors such as age, parity & booked status, the groups of cases and controls were considered as comparable groups.

Table VIII

Comparison of Gestational age of 2 groups

Gestational Age	Cases		Controls		Significance
	No	%	No	%	
28 – 30 wks	24	23.1	-	-	
30 – 37 wks	80	76.9	41	42.7	P < 0.001
38 – 40 wks	-	-	55	57.3	
Total	104	100	96	100	

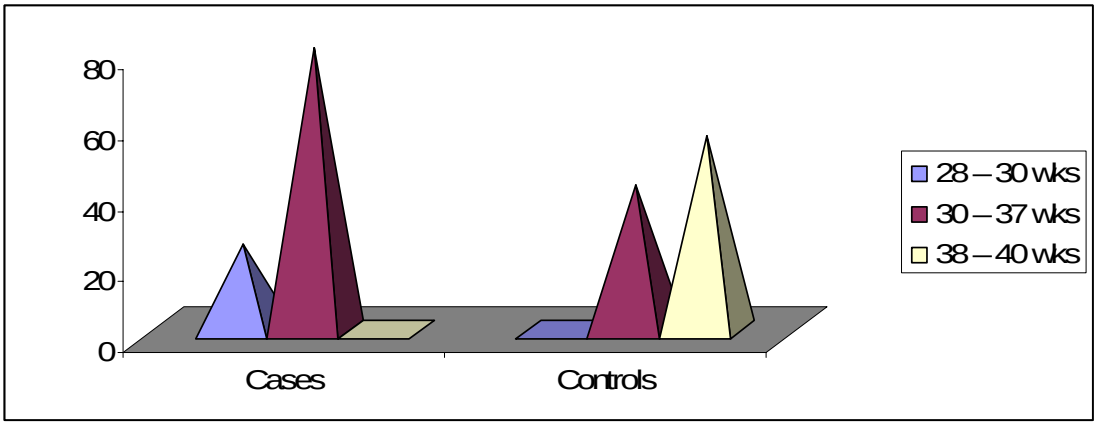
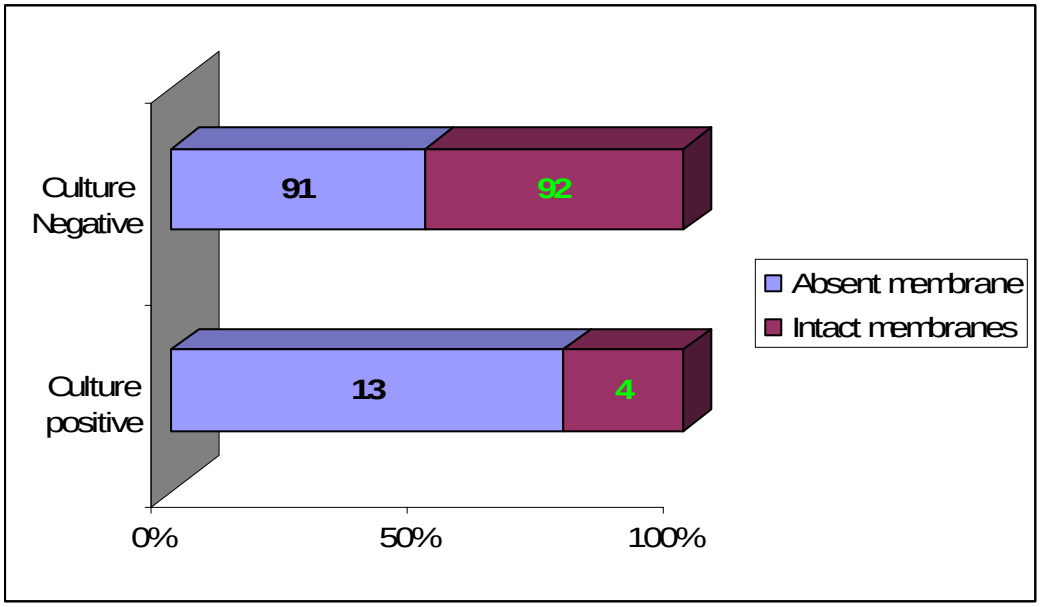


Table VIII shows the Gestation age of cases and controls. Majority of cases are between 30 – 37 wks of Gestation ie 76.9% Compared 42.7% in control group. The difference is statistically highly significant.

Table IX

IX membrane status

Membrane Status	Culture positive		Culture Negative	
	No	%	No	%
Absent membrane	13	12.5%	91	87.5%
Intact membranes	4	4.2%	92	95.8%

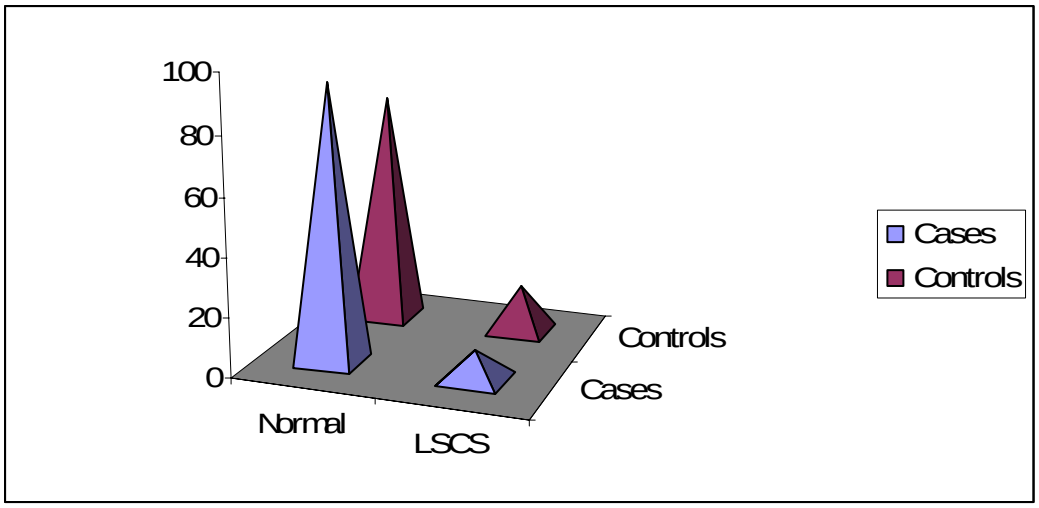


In the GBS culture positive group, 4 cases (ie) 4.2% were with intact membranes as against 13 cases (12.5%) who were with absent membranes. This is statistically highly significant with $P<0.05$.

Table X

Comparison of mode of Delivery between the groups

Gestational Age	Cases		Controls		Z	Significance
	No	%	No	%		
Normal	94	90.4	80	83.3%		
LSCS	10	9.6	16	16.7%	1.486	P < 0.001
Total	104	100%	96	100%		

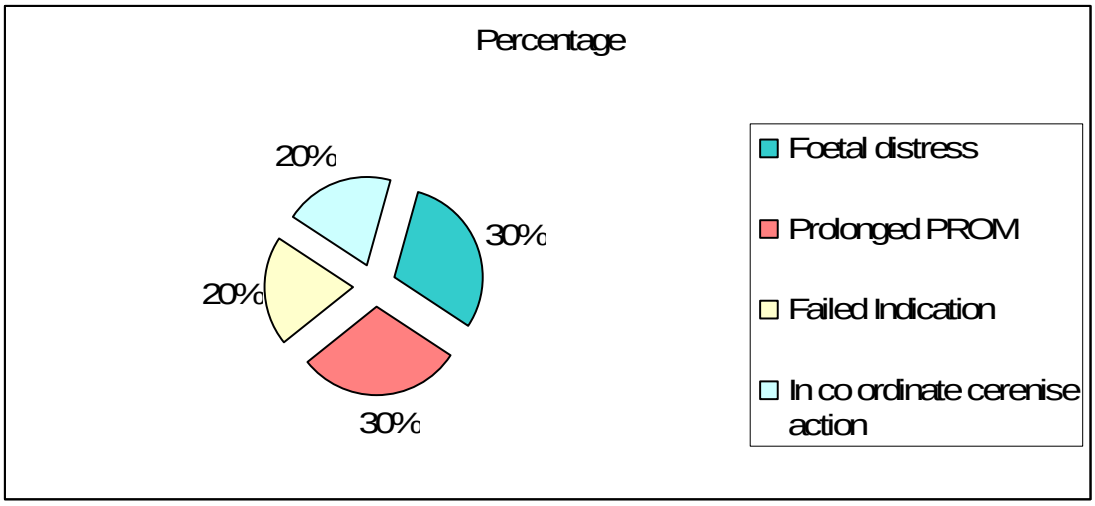


The normal deliveries among the case group was 90.4% as compared to control group which has 83.3% The difference between the 2 group was not statistically significant.

Table XI

Indications for LSCS in culture positive group

Indication	Percentage
Foetal distress	30 %
Prolonged PROM	30 %
Failed Indication	20 %
In co-ordinate uterine action	20 %



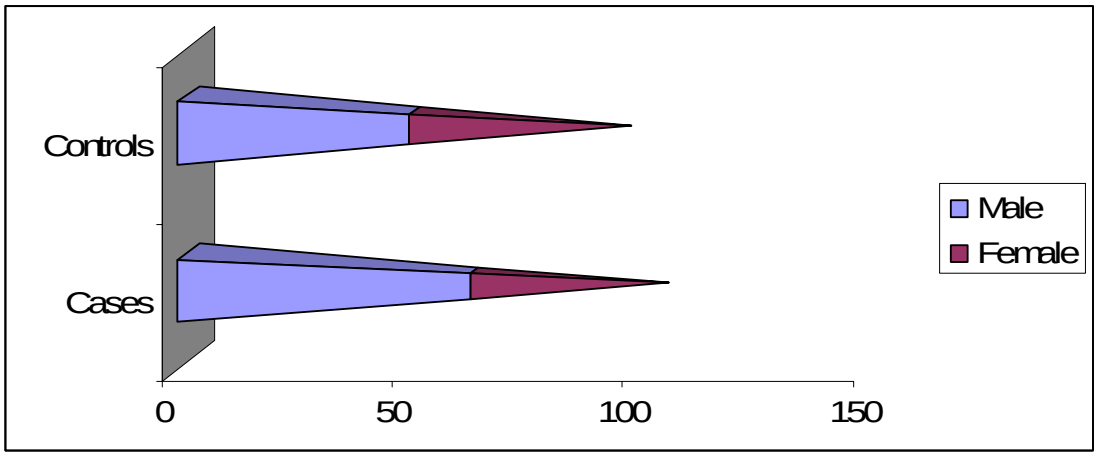
In the culture positive group 30 % had foetal distress and 30 % had prolonged PROM.

Foetal distress was in main indication for LSCS in the culture positive & culture Negative groups.

Table XII

Comparison of cases and control in respect of sex of the babies

Sex	Cases		Controls		Z	Significance
	No	%	No	%		
Male	62	59.6	49	51%		
Female	42	40.1	47	49%	1.228	P < 0.05
Total	104	100%	96	100%		



In the study group, it has been observed that out of 104 babies 59.6% were males as compared to 51% males in control group. This is not statistically significant. P > 0.05.

Table XIII shows the duration of 1st and 2nd stages of labour in the culture positive and Negative groups.

Table XIII a

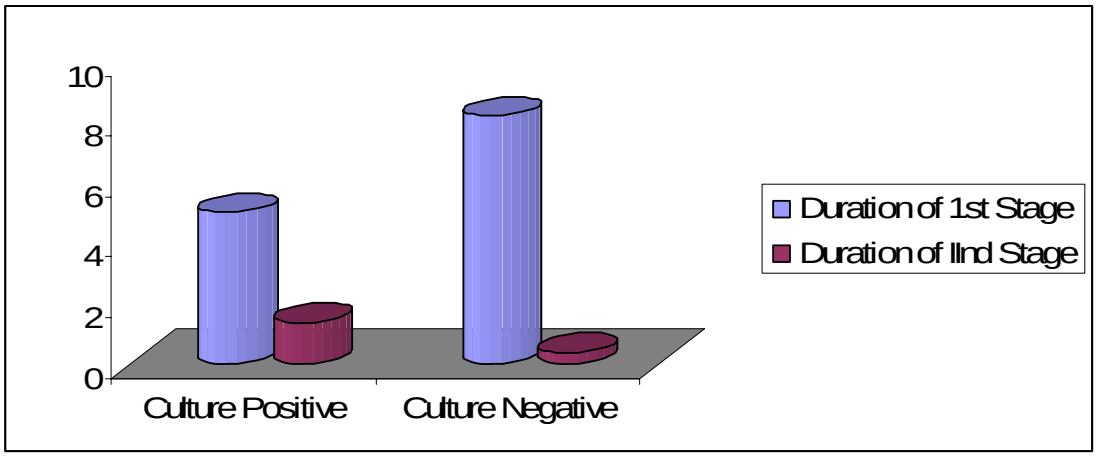
Duration of 1st Stage

Group	Duration	Average ± SD (hrs)
Culture Positive	2 – 8 hrs	8.46 ± 3.15
Culture Negative	1.3 – 15.1 hrs	6.54 ± 2.89

Table XIII b

Duration of IInd Stage

Group	Duration	Average ± SD (hrs)
Culture Positive	10 mt – 3 hrs	0.38 ± 0.36
Culture Negative	5 mt – 1.05 hrs	0.26 ± 0.13

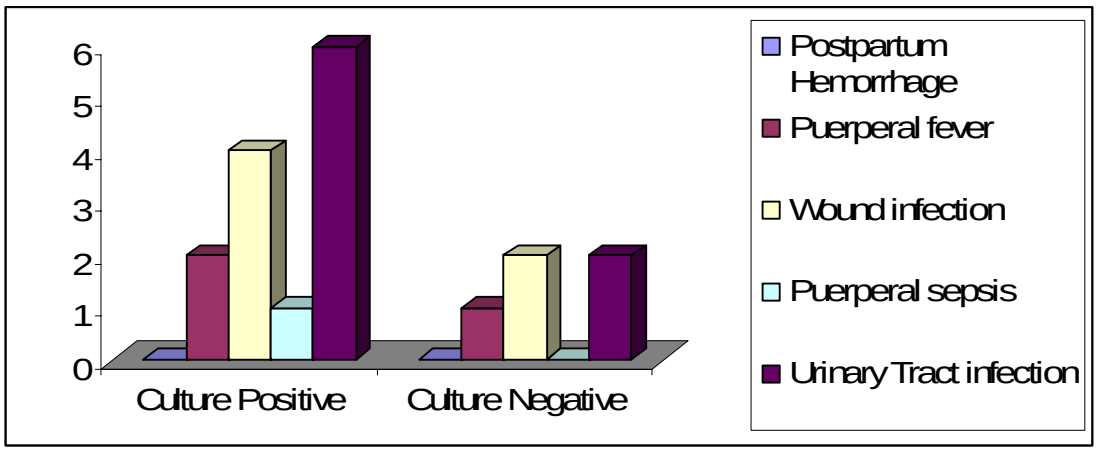


From our observation it was worked that the duration of 1st and 2nd stage were not altered in both culture positive and negative groups.

Table XIV

Maternal Morbidity

Complications	Culture Positive		Culture Negative	
	No	%	No	%
Postpartum Hemorrhage	Nil		Nil	
Puerperal fever	2	15%	1	1%
Wound infection	4	37.7%	2	2.3%
Puerperal sepsis	1	7.0%	Nil	
Urinary Tract infection	6	46.1%	2	2.1%

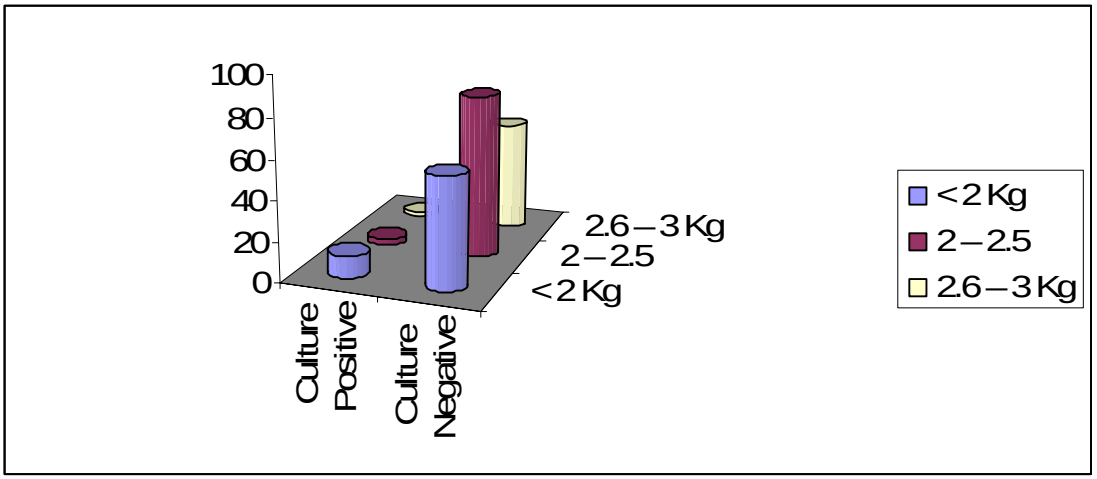


From the above data, we observed that there was an increased incidence of Puerperal fever, Wound infection, puerperal sepsis and urinary tract infection in culture positive group.

Table XV

Birth Weight of fetuses

Complications	Culture Positive		Culture Negative	
	No	%	No	%
< 2 Kg	11	61%	56	41.2%
2 – 2.5	3	18%	82	13.1%
2.6 – 3 Kg	2	12.4%	56	41.2%



In GBS positive group II cases (ie) 61% were very low birth weight where as 3 cases (ie) 18% belonged to low birth weight. 2 cases (ie) 12.4% in culture positive group weighted between 2.6 – 3 Kg.

In culture negative group very low birth babies are 41.2%. These difference in satisfactory significant with P values < 0.05.

Table XVI

Comparison of morbidity of neonates between the cases and control group

Morbidity of Neonates	Cases		Controls		Z	Significance
	No	%	No	%		
Present	17	16.3	1	1%	4.067	P < 0.001
Absent	87	83.7	95	99%		
Total	104	100%	96	100%		

The difference between the two groups was statistically highly significant P<0.001.

Correlation of Neonatal's culture positive with Mother's culture positive – between the groups

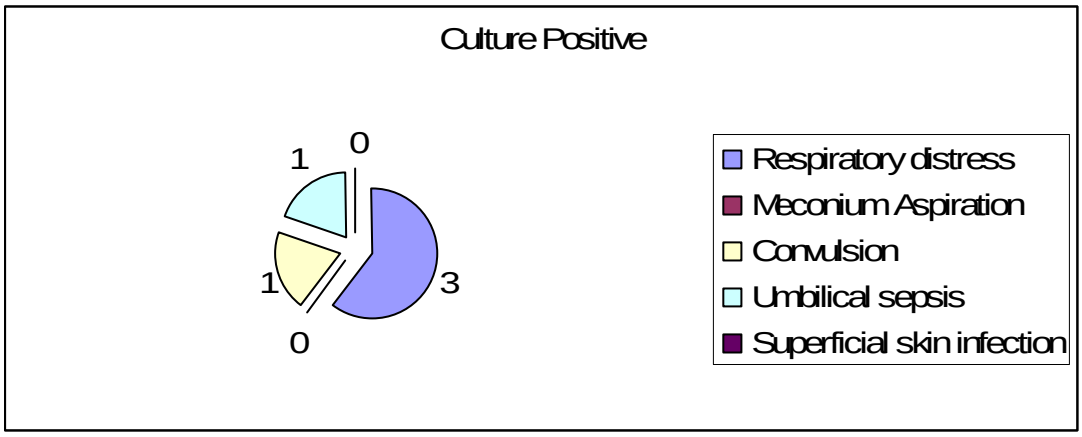
Neonatal GBS	Cases Group Mother's GBS				Control group Mother's GBS				Total Mother's GBS				Total	
	+ve		-ve		+ve		-ve		+ve		-ve			
	No	%	No	%	No	%	No	%	No	%	No	%	No	%
Positive	5	38.5	0	0	0	0	1	1	5	29.4	1	0.5	6	3
Negative	8	61.5	91	100	4	100	91	99	12	70.6	182	99.5	194	97
Total	13	100	91	100	4	100	92	100	17	100	183	100	200	100
ψ^2	36.768				0.044				44.538				-	
d.f	1				1				1					
Significance	P<0.001				P>0.05				P<0.01					

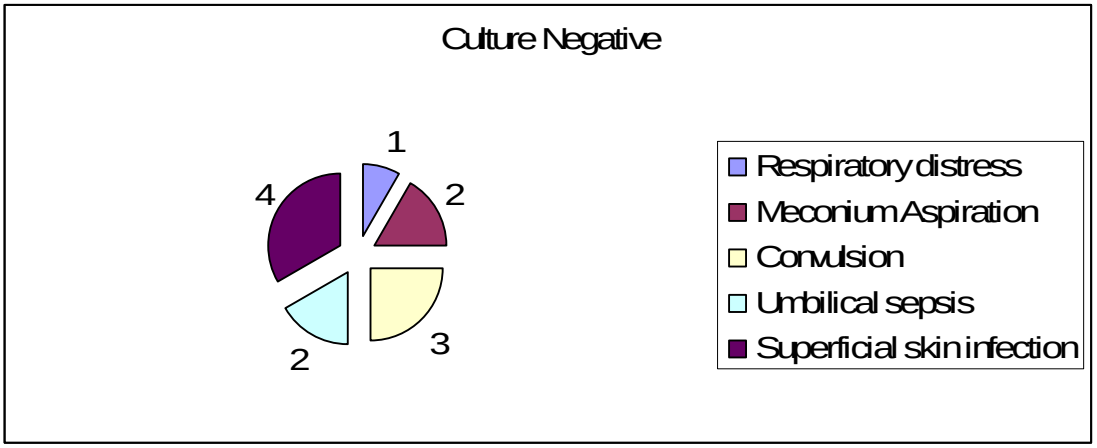
This table shows the number of culture positive neonates is high in culture positive mother's.

Table XVII

Neonatal Morbidity in GBS positive group

Problems	Culture Positive		Culture Negative	
	No	%	No	%
Respiratory distress	3	37%	1	1%
Meconium Aspiration	-	-	2	2.2%
Convulsion	1	12.5%	3	3.0%
Umbilical sepsis	1	12.5%	2	2.2%
Superficial skin infection	Nil		4	4.4%





From the above data we observed that Respiratory distress syndrome in 3 cases (ie) 37% as compared to 1% in culture negative group.

Convulsion in 12.5% cases in culture positive group as compared to 3% in culture negative group.

Umbilical sepsis in 12.5% cases in culture positive group as compared to 2.2% in culture negative group.

DISCUSSION

Infectious organisms play an important role in the occurrence of preterm birth. Whether Group B Streptococci are also a cause of preterm delivery has been the subject of conflicting reports.

The largest controlled study of this issue in a cohort of 13,646 pregnant women conducted as part of the vaginal Infections and prematurity study found that women heavily colorized with Group B Streptococci at 23 – 26 weeks gestation were most likely to delivery a preterm, low birth weight infant and that those heavily colonized at the time of delivery were more likely to delivery prematurely (Regan et al 1991)

Infection of the maternal genital tract plays a major role directly or indirectly in causation of premature rupture of membranes which is associated with maternal morbidity, increased neonatal morbidity and even mortality.

Though clinically inapparent, infection is shown to be associated with approximately 30% of cases of premature rupture of membrane especially preterm (Miller et al 1978 & Johnson et al 1981)

The organisms like N. Gonorrhoea Group B Streptococci have been associated with preterm labour (Regan et al 1981; Monikoff et al 1984 Mc Gregar et al 1987) had demonstrated that protease production by both are acknowledged pathogenic and commensal beacteria may

contribute to the occurrence of reproductive tract morbidity including premature rupture of membranes and preterm labour.

There is a conflicting evidence regarding a possible causal role for Group B Streptococci in the development of preterm premature rupture of membranes. The relative prevalence of endocervical infection with Group B Streptococci in patients with preterm premature rupture of membranes was compared with a control group taken from the same obstetric population. Group B Streptococci were isolated from 16% of the patients with preterm premature rupture of membranes Vs 4% of the control population ($P<0.05$) AJOG 1988.

In a prospective study at a single hospital in Odense, Denmark endocervical culture where obtained at delivery from women with preterm delivery and from a random sample of women delivering at term. 12 out of 84 (14%) of women with preterm delivery were clonised at delivery with Group B Streptococci than women with term deliveries (22/300, 7%) AJOG 22001; 184: 427 – 33; Daniel et al; 1992.

In our present study Group B Streptococci was isolated from 13 out of 104 (12.5%) with premature rupture of membranes Vs 4 out of 96 cases (4.2%) in control population with P value < 0.05.

Colonisation with Group B Streptococci at the time of delivery was positively associated with Group B Streptococci at delivery were 3 times were likely to have a preterm delivery than were women who were not

colonized. These results corroborate the findings of the vaginal interim and prematurity study.

Although some studies of Group B Streptococci colnization at delivery showed an association with preterm delivery, other studies did not.

Baker et al	PTL	11%	6.3%
	PROM	11%	3.8%
Regan et al	PTL	5.4%	1.26%
	PROM	15.3%	7.0%
Minkoff et al	PTL	17.1%	8.7%
	PROM	10.0%	8.8%
Hastings et al	PTL	6.5%	6.4%
Boh et al	PROM	5.6%	3.4%
	LBN	8.4%	1.7%
Lamon et al	PTL	6.0%	-

The only clearly positive study is that of Regan et al. The results of Bobih et al are also supportive of this view.

Age :

In a study of association of Group B Streptococci with preterm premature rupture of membranes by Alger et al 1983, it was found that

the risk of preterm premature rupture of membranes and Group B Streptococci was higher in younger patients.

In our present study it was found that the risk of preterm rupture of membranes associated with Group B Streptococci infection was markedly increased in older age group with median age of 25 compared to Age 23 of control group.

Antenatal care

Slightly increased incidence of Group B Streptococci in unbooked cases compared to booked case. The lower incidence in booked cases may be attributed to early detection and treatment of vaginal infection.

Maternal Morbidity

The major maternal complication of Group B Streptococci are chorio amnionitis, Sepsis, and urinary tract infection.

In the present study no case of chorio amnionitis were reported in Group B Streptococci positive group. The incidence of this complication varies from 3 to 31% (Gibbs et al; 1989 Kvalason et al 1980; Moral et al; 1989, Dale et al 1989)

The absence of chorio amnionitis with present study may be due to routine use of prophylactic antibiotic in all cases of premature rupture of membranes.

In this study the incidence of febrile morbidity was 15%, wound infection 37.7% and Urinary tract infection 46% in the culture positive group.

Neonatal morbidity

Neonatal morbidity and mortality secondary to Group B Streptococci are strongly affected by the gestational age at the time of delivery (Taylor et al; 1984l; Vaille et al 1988). During a 10 year study between 1988 – 1997 in Vellore, only 10 cases of Neonatal Group B Streptococci infections were identified.

This gives the incidence of 0.17 per 1000 live births. However this member represents only the cases occurring among deliveries in a tertiary care centre.

In our study the incidence of respiratory distress 37% and umbilical Sepsis 12.5%. The major risk to the foetus is that of complication of prematurity rather than sepsis.

NEONATAL MORTALITY IN STUDY GROUP

One Neonatal death occurred in culture positive group in this study due to respiratory distress. But Blood culture is negative for bacterial sepsis.

Daikathu et al – 1991 of the opinion that neonatal infections were due to prematurity rather than premature rupture of membranes Lebherg and Autin reported that prematurity alone is the cause of perinatal deaths in over 50% of cases.

Relation ship between vaginal colonization with other microorganism and Group B Streptococci.

Our results support a previous finding that a deficit in Lactobacillus may allow a colonization with Group B Streptococci. More over Group B Streptococci appeared to be an opportunistic colonizer (Danieal et al, Thorsel et al 1992)

However our study had certain limitations. The density of vaginal colonization may be important in influencing preterm delivery. The vaginal infection and prematurity study showed that only heavy colonization, defined as growths in non selective media, was associated with preterm delivery. In our study there was an admitter of heavy and light colonization with Group B Streptococci.

SUMMARY AND CONCLUSION

During the study period, 104 cases of premature rupture of membranes and 96 controls of Term Gestation with intact membranes were studied. The association between Group B Streptococci and preterm premature rupture of membranes was Analysed. Also the effect of Group B Streptococci positive in pregnancy outcome was Analysed.

12.5% was colonized by Group B Streptococci in the study group as against 4.2% in the control group. The mean age in the cases was 25 years as compared to 23 years in the control group. The older patients were affected more in this study.

Evidence of Group B Streptococci in unbooked cases was slightly high in the present study.

The mode of delivery (ie) vaginal delivery Vs LSCS is not altered significantly in this study. Vaginal deliveries were more in the culture positive group.

Duration of 1st and 2nd stages were not altered by culture positivity in those cases who delivered vaginally. As regards to birth weight of foetuses in the culture positive cases, 61% were below 2 kg. In culture negative group only 41.2% were below 2 kg.

Coming to the maternal morbidity, commonest maternal morbidity was urinary tract infections in 46.1% cases, wound infection in

37.7% cases and puerperal fever in 15% cases and puerperal sepsis in 7% cases. There was no maternal deaths in either groups.

Neonatal morbidity commonest cause is respiratory distress - 37%, convulsion 12.5% and umbilical sepsis 12.5% in culture positive group.

Neonatal morbidity – 1 neonatal death occurred in this study due to respiration distress

CONCLUSION

In conclusion, we have found an association that cervical infection with Group B Streptococci at delivery remained a risk factor for preterm delivery.

Also Group B Streptococci may be a leading cause of both neonatal and puerperal infection. In 1996, the centre for diseases control and preveution (CDC) published guidelines for the prevention of perinatal Group B Streptococci disease, which included a culture based screening strategy for the identification and subsequent intrapartum prophylaxis of those women colonized with Group B Streptococci. With the advent of these guidelines however the incidence of perinatal Group B Streptococci disease has decreased in the past few years.

Also, disruption of the normal vaginal flora predominated by Lactobacilli, may allow for colonization with pathogenic bacteria that play a role in preterm delivery. More over Group B Streptococci appeared to be an oppurtunisitic colonizer, more likely to be present in the absence of normal flora, but also in the absence of other abnormal flora such as G. Vaginalis.

Thus the establishment and maintenance of normal vaginal flora in pregnant women may be the way of reducing the risk of premature rupture of membrane and preterm delivery.

PREVENTION

With the advent of consensus guidelines, the incidence of perinatal Group B Streptococci disease has decreased in the past few years.

- Colonisation with Group B Streptococci however, may lead to other undesired peripartum events, such as preterm delivery, that are not addressed by these guidelines.
- Several prevention strategies might be employed screening methods for early detection, then an attempt to eradicate Group B Streptococci from the genital tract in the antepartum period.

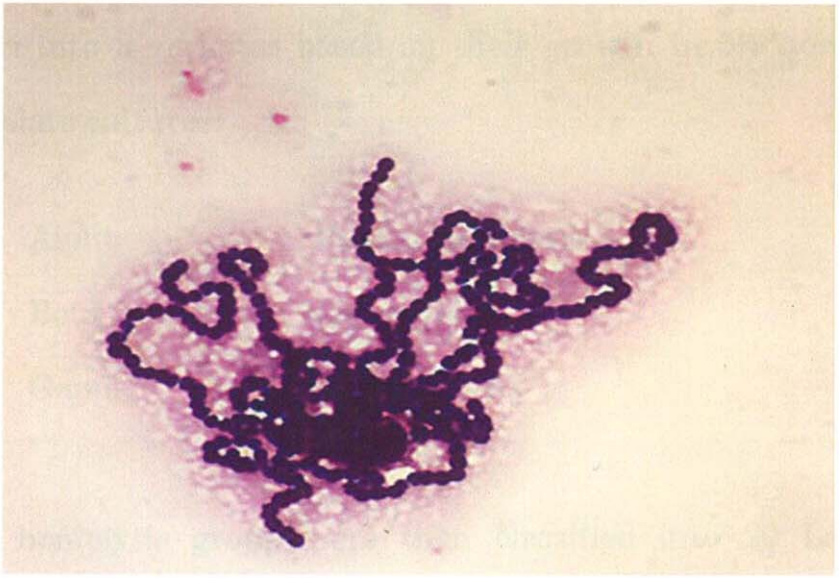
A second preventive strategy would involve the use of intrapartum antibiotic prophylaxis. This would shorten antibiotic exposure time for both mother and the foetus.

It has been reported that invasive Group B Streptococci infection declined by 21% in pregnant women, suggesting that intrapartum prophylaxis, protects the mother as well as the neonate (AJOG Nov 2001).

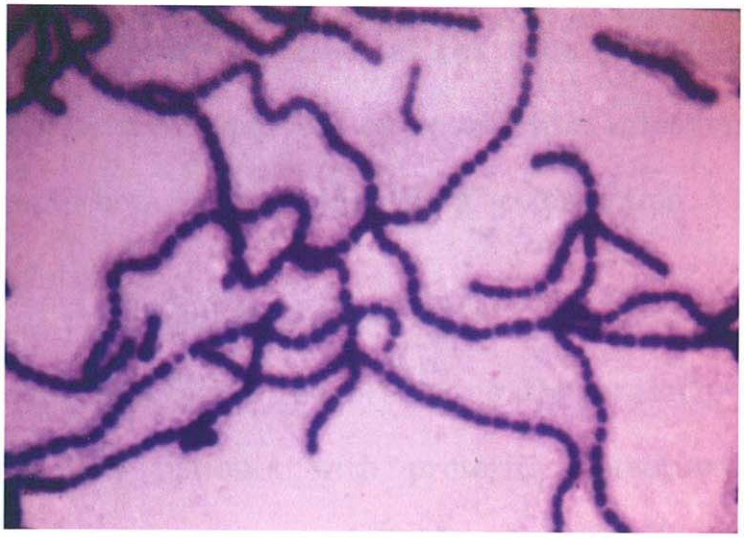
- A third preventive strategy could involve the use of both antepartum eradication and intrapartum prophylxis. Prevention of infection will decrease the fetal morbidity and may prolong the pregnancy and increase fetal survival.

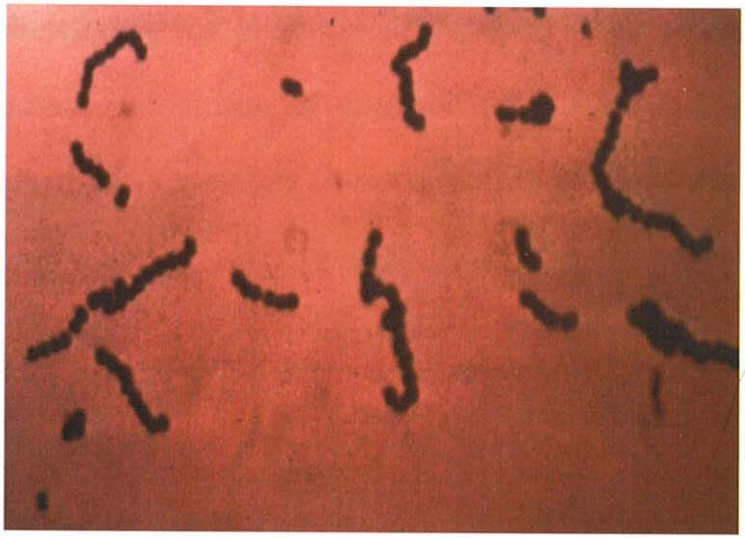
- Vaccines trail is going on, though trials in pregnant women would be ethically difficult and large sample sizes would be needed to show an effect on neonatal sepsis rates. The vaccines strategies involved conjugating capsular polysaccharide such as C_{5a} peptidase with T-cell dependant protein antigens.

STREPTOCOCCUS AGALACTIAE

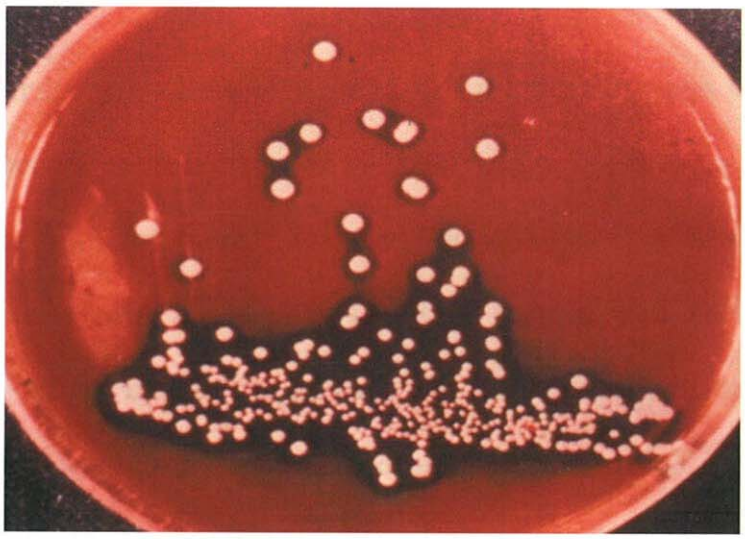


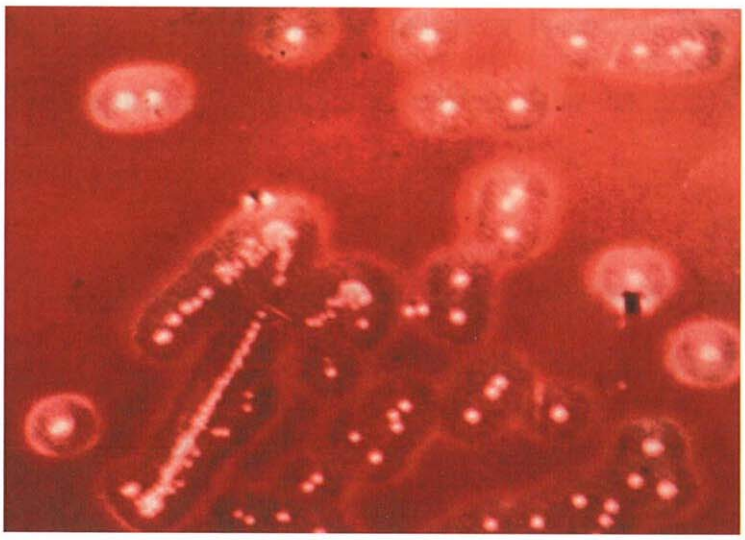
GROUP B STREPTOCOCCI
GRAM STAIN





BETA HEMOLYTIC STREPTOCOCCI





PRETERM BABY WITH RESPIRATORY DISTRESS

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PROFORMA

Name

Age

I.P. NO

Occupation

Education

SE Status

Marital Status Para Live

Abortion

LMP : EDD

Booked / Unbooked :

Immunisation : Yes No

D.O.A. D.O. Delivery

D.O. Discharge

Presenting Complaints

1. Period of gestation
2. Presence of pains
3. Draining p/v
4. Duration of draining p/v
5. H/o. fever
6. H/o four smelling discharge p/v
7. H/o burning micturition

AN History

I Trimester
II Trimester
III Trimester

Past History

Menstrual History

Obstetric History : Previous H/o preterm birth

Previous H/o abortion

Previous H/o prom

Personal History :

Family History :

Examination

General

1. Height
2. Weight
3. Temperature
4. Pallor
5. Icterus
6. Pedal aedema
7. Pulse rate
8. BP
9. Spine
10. Thyroid

Obstetrical Exam :

P/A :

S/E :

P/V :

Investigation

Routine	1.	Hb
	2.	Blood grouping and Rh typing
	3.	Urine routine
	4.	Total leukocyte count
	5.	Differential leukocyte count
	6.	HIV
	7.	VDRL
Specific	8.	Culture

Sample Collected

Mother	***Baby***
1. Sample from vaginal Pool	Skin surface swab
2. Anorectal Swab	Ear swab
3. Urine	

Labour

Duration : Any complications

1st Stage

2nd Stage

3rd Stage

Baby

a) Live born / Still born :

b) Apgar 1’ :

5’ :

10’ :

c) Gestational age :

d) Weight :

e) Sex :

f) Whether asphyxiated

If so type of resuscitation :

Maternal Morbidity

a. Postpartum haemorrhage
b. Puerperal fever
c. Wound infection
d. Puerperal sepsis
e. Urinary tract infection

Fetal morbidity

a) Respiratory Distress syndrome
b) Meconium aspiration
c) Convulsion
d) Umbilical sepsis
e) Superficial skin infection

Maternal Mortality

Cause

Neonatal Mortality

Cause

Condition at discharge

Mother

Baby

MASTER CHART

Sl. No	IP No:	Name	Age in years	Booked (1) / UnBooked (2)	Parity	Gestational Age in weeks	Status of Membranes Present 1 Absent 2	Mother culture Positive 1 Negative 2	Mode of Delivery Normal-1 Caesarean-2	Baby			Neonatal Outcome Morbidity Present 1 Absent 2	Post Partum Complications Present 1 Absent 2
										Sex Male-1 Female-2	Weight in Kg.	Culture Positive 1 Negative 2		
1	23305	Chandra	35	1	1	35	2	2	1	1	2.0	2	2	2
2	49315	Kalaimathi	28	1	1	34	2	2	2	1	1.9	2	2	2
3	23769	Santhi	30	2	2	35	2	2	1	2	1.6	2	1	2
4	49571	Muthumani	28	1	1	40	1	2	1	1	3.0	2	2	2
5	48948	Saraswathy	24	1	1	38	1	2	1	2	2.5	2	2	2
6	49183	Mahalakshmi	23	1	1	40	1	2	2	1	3.2	2	2	2
7	24338	Valli	37	2	2	32	2	1	1	2	1.1	2	1	1
8	23632	Gomathy	27	1	1	38	1	2	1	2	3.0	2	2	2
9	25268	Shanu	19	2	1	35	2	2	1	2	1.4	2	1	2
10	49240	Petchiammal	20	1	1	36	2	2	1	1	1.9	2	2	2
11	25386	Mariammal	39	2	2	34	2	1	1	1	1.2	1	1	2
12	28246	Vijayalakshmi	34	1	1	36	2	2	1	2	1.7	2	2	2
13	48956	Menaga	23	1	2	38	1	2	1	1	2.8	2	2	2
14	48634	Petchithai	25	1	1	37	1	2	2	2	2.5	2	2	2
15	49000	Rajakani	24	1	1	36	1	2	2	2	3.0	2	2	2
16	29317	Veerasundari	27	2	2	35	2	2	1	1	1.6	2	2	2
17	48581	Bhavani	27	1	1	36	2	2	1	1	2.0	2	2	2
18	29503	Essakkiammal	25	1	1	37	2	2	2	1	1.9	2	2	2
19	50735	Noor Fathima	23	1	1	35	2	1	1	2	1.4	1	1	1
20	48814	Vijayalakshmi	27	1	2	38	1	2	1	2	2.7	2	2	2
21	48973	Mariammal	30	1	2	39	1	2	1	1	2.7	2	2	2
22	46222	Sudali	25	1	2	37	1	2	1	2	2.4	2	2	2
23	29945	Sakthi Kani	25	1	2	38	1	2	1	2	2.4	2	2	2
24	49164	Palavesi	24	1	1	40	1	2	2	1	3.4	2	2	2
25	40052	Muthulakshmi	23	2	1	30	2	1	1	1	1.0	1	1	2
26	50731	Selvi	32	1	1	36	1	2	1	2	2.5	2	2	2
27	48730	Petchithai	23	1	2	38	1	2	2	1	2.7	2	2	2
28	49695	Gomathy	21	1	1	35	1	2	1	1	2.5	2	2	2

Sl. No	IP No:	Name	Age in years	Booked (1) / UnBooked (2)	Parity	Gestational Age in weeks	Status of Membranes Present 1 Absent 2	Mother culture Positive 1 Negative 2	Mode of Delivery Normal-1 Caesarean-2	Baby			Neonatal Outcome Morbidity Present 1 Absent 2	Post Partum Complications Present 1 Absent 2
										Sex Male-1 Female-2	Weight in Kg.	Culture Positive 1 Negative 2		
29	49681	Sakthi Kani	27	1	1	34	2	2	1	1	1.9	2	2	2
30	49726	Prema	22	1	1	32	2	1	1	1	1.2	2	1	2
31	39735	Amudha	35	2	2	36	2	2	1	1	2.0	2	2	2
32	47654	Indirani	25	1	2	37	2	2	2	1	2.8	2	2	2
33	49094	Suguna	24	1	2	37	1	2	2	1	3.8	2	2	2
34	47670	Muthuselvi	24	1	1	38	1	2	1	1	2.2	2	2	2
35	47371	Selvi	30	1	2	38	1	2	2	2	2.6	2	2	2
36	49133	Essakkiammal	23	1	1	34	2	2	1	2	1.3	2	1	2
37	48943	Ramalakshmi	25	1	2	36	2	2	2	1	2.0	2	2	2
38	48275	Shenbhagavalli	21	1	1	35	2	2	1	2	1.7	2	2	2
39	49237	Saraswathy	22	1	1	38	1	2	1	2	3.2	2	2	1
40	50431	Meena	27	1	2	34	1	2	1	2	1.7	2	2	2
41	46562	Valliammal	22	1	1	34	2	2	1	2	1.5	2	1	2
42	32134	Sudali	23	2	2	38	1	2	2	1	2.5	2	2	2
43	48465	Rishu Fatima	25	1	2	37	1	2	2	1	3.1	2	2	1
44	48590	Murugammal	23	1	2	38	1	2	1	1	2.0	2	2	2
45	50799	Parvathy	29	2	2	36	2	2	1	2	1.5	2	2	2
46	50066	Mariammal	25	1	1	35	2	2	1	2	1.4	2	1	2
47	53258	Mala	33	2	2	35	2	1	1	1	1.4	2	1	2
48	49219	Priya	24	1	2	36	2	2	1	2	2.1	2	2	2
49	50178	Selvi	28	1	1	36	1	2	1	2	2.2	2	2	2
50	48662	Muthumari	20	1	1	37	1	2	1	2	2.6	2	2	2
51	49826	Paulkani	21	1	2	38	1	2	2	2	2.0	2	2	2
52	53285	Vellathai	24	2	1	34	2	1	1	1	1.0	1	1	2
53	49493	Thirumalai selvi	30	1	1	37	1	2	1	2	2.1	2	2	2
54	31796	Vellathai	25	2	1	32	2	1	1	2	1.0	2	1	2
55	49488	Devakirubai	29	1	1	36	1	2	1	1	2.7	2	2	2
56	50438	Lakshmi	22	1	2	35	1	2	1	2	2.25	2	2	2
57	44637	Gnanasoundari	36	1	2	30	2	2	1	1	2.5	2	2	2

Sl. No	IP No:	Name	Age in years	Booked (1) / UnBooked (2)	Parity	Gestational Age in weeks	Status of Membranes Present 1 Absent 2	Mother culture Positive 1 Negative 2	Mode of Delivery Normal-1 Caesarean-2	Baby			Neonatal Outcome Morbidity Present 1 Absent 2	Post Partum Complications Present 1 Absent 2
										Sex Male-1 Female-2	Weight in Kg.	Culture Positive 1 Negative 2		
58	50203	Shepa	28	1	1	28	2	2	1	1	2.5	2	2	2
59	50127	Avudaiyatchi	25	1	1	36	2	2	1	1	1.5	2	2	2
60	52177	Mariammal	37	1	1	35	2	2	1	2	1.5	2	2	2
61	49727	Chandra	23	1	2	36	2	2	1	1	1.7	2	2	2
62	31316	Utchimahali	35	1	1	35	2	2	2	1	1.5	2	2	2
63	50173	Petchiammal	21	1	2	35	2	2	1	2	2.1	2	2	2
64	50673	Mala	28	1	1	34	2	2	1	2	1.9	2	2	2
65	49029	Ramalakshmi	30	1	2	32	2	1	1	1	1.0	1	1	1
66	50179	Rajeswari	25	1	2	38	1	2	1	2	2.9	2	2	2
67	30407	Mariammal	25	2	1	37	1	2	1	1	2.0	2	2	2
68	49673	Prema	30	1	1	40	1	2	2	2	2.7	2	2	1
69	49210	Padma	23	1	2	40	1	2	1	2	4.2	2	2	2
70	51012	muthumari	22	1	2	36	1	2	1	2	1.7	2	2	2
71	50814	Chitraimari	25	1	2	35	2	2	1	1	1.7	2	2	2
72	39945	Ramalakshmi	23	2	2	35	2	2	1	2	1.7	2	2	2
73	47541	Sudha	23	1	2	37	2	2	1	1	2.0	2	2	2
74	49969	Ambigha	21	1	2	29	2	2	1	1	2.5	2	2	2
75	51005	Sumathy	30	1	2	37	2	2	2	1	1.8	2	2	2
76	50180	Geetha	26	1	1	34	2	2	1	2	2.2	2	2	2
77	32202	Muthulakshmi	29	1	2	37	2	2	1	1	2.0	2	2	2
78	30407	Mariammal	22	1	1	35	2	2	1	2	1.5	2	2	2
79	36969	Ramyala Begum	36	1	2	37	2	2	1	1	2.0	2	2	2
80	50097	Rajapushpam	26	1	2	35	2	2	2	2	1.9	2	2	2
81	39890	Parvathy	30	1	1	34	1	2	1	2	1.6	2	2	2
82	36154	Subbulakhsmi	30	1	2	38	1	2	2	1	3.2	2	2	2
83	50706	Mariammal	21	1	2	39	1	2	1	2	2.7	2	2	2
84	49924	Mydeen Beevi	20	1	1	40	1	2	1	2	3.0	2	2	2
85	34814	Mariammal	24	2	1	36	1	2	1	2	2.4	2	2	2
86	49925	Valliammal	23	1	1	38	1	2	1	2	2.7	2	2	2

Sl. No	IP No:	Name	Age in years	Booked (1) / UnBooked (2)	Parity	Gestational Age in weeks	Status of Membranes Present 1 Absent 2	Mother culture Positive 1 Negative 2	Mode of Delivery Normal-1 Caesarean-2	Baby			Neonatal Outcome Morbidity Present 1 Absent 2	Post Partum Complications Present 1 Absent 2
										Sex Male-1 Female-2	Weight in Kg.	Culture Positive 1 Negative 2		
87	50348	Mariammal	23	1	2	40	1	2	1	1	2.6	2	2	2
88	33943	Annamary	24	2	2	40	1	2	2	2	3.2	2	2	1
89	50078	Pitchi Rose	23	1	1	37	1	2	1	2	2.0	2	2	2
90	49772	Victoria	24	1	1	35	1	2	1	1	2.5	2	2	2
91	51023	Ksiammal	21	1	1	36	1	2	1	1	2.5	2	2	2
92	50982	Subasankari	21	1	1	37	1	2	1	1	2.0	2	2	2
93	32922	Periathi	25	2	1	38	1	2	1	1	2.0	2	2	2
94	57002	Rajathi	21	1	1	40	1	2	1	1	2.6	2	2	2
95	39591	Geetha	23	2	2	38	1	2	1	2	2.5	2	2	2
96	50182	Santhya	20	1	1	38	1	2	1	1	3.2	2	2	2
97	36169	Prema	25	2	2	40	1	2	2	2	3.0	2	2	1
98	40570	Subbulakshmi	23	1	1	35	2	2	1	1	1.8	2	2	2
99	50195	Karthikai kani	20	1	2	36	2	2	1	2	1.6	2	2	2
100	50855	Radha	28	1	2	35	2	2	1	1	1.5	2	1	2
101	40660	Usha	30	1	1	35	2	2	1	1	1.75	2	2	2
102	48079	Mookammal	34	1	1	37	2	2	1	1	2.0	2	2	2
103	47614	Narzeena Begum	30	1	2	28	2	2	1	1	2.0	2	2	2
104	36969	Ramalakshmi	22	1	2	37	2	2	1	2	2.0	2	2	2
105	50643	Ananthi	20	1	1	34	2	2	1	2	2.1	2	2	2
106	25089	Pushaparadha	20	2	1	28	2	2	1	1	2.0	2	2	2
107	36059	Rani	22	1	2	30	2	2	1	2	2.1	2	2	1
108	36797	Shathi	29	1	1	29	2	2	1	1	2.0	2	2	2
109	50361	Subbulakshmi	25	1	1	34	2	1	2	1	1.2	2	1	1
110	49897	Sudali	19	1	1	37	1	2	1	2	2.2	2	2	2
111	47413	Banu	22	1	1	36	1	2	1	1	3.0	2	2	2
112	38567	Sowbagya	27	1	1	38	1	2	1	1	3.0	2	2	2
113	42378	Mahaeswari	23	1	1	40	1	2	1	2	3.2	2	2	2
114	53156	Kaleeswari	25	1	2	39	1	2	1	2	3.0	2	2	2
115	43976	Pappathy	25	1	1	38	1	2	1	1	2.5	2	2	2

Sl. No	IP No:	Name	Age in years	Booked (1) / UnBooked (2)	Parity	Gestational Age in weeks	Status of Membranes Present 1 Absent 2	Mother culture Positive 1 Negative 2	Mode of Delivery Normal-1 Caesarean-2	Baby			Neonatal Outcome Morbidity Present 1 Absent 2	Post Partum Complications Present 1 Absent 2
										Sex Male-1 Female-2	Weight in Kg.	Culture Positive 1 Negative 2		
116	23920	Valliammal	28	1	2	38	1	2	1	2	2.2	2	2	1
117	32002	Petchiammal	21	2	1	37	1	1	1	1	3.0	2	2	2
118	49345	Shanthi	25	1	2	36	1	2	1	2	2.7	2	2	2
119	50195	Santhanamari	20	1	1	37	1	2	1	1	2.5	2	2	2
120	48049	Malathy	20	1	1	38	1	2	1	1	2.0	2	2	2
121	50984	Maragatham	21	1	2	40	1	2	1	1	3.0	2	2	2
122	52928	Meghala	25	1	1	39	1	2	1	2	2.5	2	2	2
123	36750	Malathy	28	1	1	35	2	2	1	1	1.8	2	2	2
124	49736	Saraswathy	24	1	1	36	2	2	1	2	1.8	2	2	2
125	48249	Jothimani Duratchi	20	1	1	37	2	2	1	2	2.2	2	2	2
126	52832	Annapappathi	23	1	1	30	2	2	1	1	1.8	2	2	2
127	49417	Muthulakshmi	24	1	1	34	2	2	1	2	1.7	2	2	2
128	53442	Lakshmi	29	1	1	36	2	2	1	2	2.5	2	2	1
129	50836	Sankarammal	21	1	2	28	2	1	2	1	1.2	2	1	1
130	49352	Vidhyavathy	25	1	1	29	2	2	1	1	2.0	2	2	2
131	49054	Rani	29	1	2	34	2	2	1	1	1.6	2	2	2
132	33259	Kuttiammal	23	1	1	30	2	2	1	2	2.5	2	2	2
133	31991	Gomathy	33	1	1	36	2	2	1	1	2.5	2	2	2
134	53554	Annamaria	42	2	2	28	2	2	1	2	3.0	2	2	2
135	50422	Sornaselvi	24	1	2	37	2	2	1	1	3.5	2	2	2
136	53348	Chinnathai	23	1	1	37	1	2	1	2	2.7	2	2	2
137	50162	Shantha	28	1	2	38	1	2	1	2	2.2	2	2	2
138	53113	Kanmani	22	1	1	36	1	1	1	1	3.0	2	2	1
139	50816	Padmavathy	26	1	1	34	2	2	1	1	1.7	2	2	2
140	50615	Chellammal	19	1	1	36	2	2	1	2	2.2	2	2	2
141	47967	Kamalam	27	1	2	35	2	2	1	1	1.8	2	2	2
142	53309	Latha	20	1	1	28	2	2	1	2	2.5	2	2	2
143	50627	Benazir Begam	19	1	1	30	2	2	1	2	3.2	2	2	2
144	50751	Soosaimary	23	1	1	28	2	2	1	2	3.0	2	2	2

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										Sex Male-1 Female-2	Weight in Kg.	Culture Positive 1 Negative 2		
145	4855	Mariammal	22	1	1	29	2	2	1	1	3.0	2	2	2
146	50954	Ambigha	19	1	1	28	2	2	1	2	3.0	2	2	2
147	51032	Selvi	21	1	1	37	2	2	1	2	2.5	2	2	2
148	51198	Gandhi	21	1	1	28	2	2	1	2	3.2	2	2	2
149	51028	Mutharasi	19	1	1	40	1	1	1	1	3.0	2	2	1
150	35240	Subbulakshmi	23	1	1	39	1	2	1	1	2.1	2	2	2
151	39829	Thangaselvi	28	1	2	36	1	2	1	1	2.8	2	2	2
152	53658	Padma	29	1	2	37	1	2	1	1	2.4	2	2	2
153	48749	Pudhiaval	31	1	2	38	1	2	1	2	2.3	2	2	2
154	51022	Paulkani	26	1	2	36	1	2	1	1	2.2	2	2	2
155	51155	Murugammal	21	1	1	35	1	1	1	1	2.3	2	2	2
156	50669	Sivagami	21	1	1	34	1	2	1	2	1.6	2	2	2
157	51123	Mari	21	1	1	40	1	2	1	1	3.0	2	2	2
158	34860	Ramalakshmi	21	1	1	37	1	2	1	2	2.9	2	2	2
159	50457	Mahalakshmi	19	2	2	36	1	2	1	2	1.2	1	1	2
160	39751	Punitha Sheela	21	1	1	38	1	2	1	1	2.7	2	2	2
161	50533	Muppidathy	31	1	2	38	1	2	1	1	2.0	2	2	2
162	52732	Thangamari	20	1	1	40	1	2	1	2	3.0	2	2	2
163	52863	Mariammal	22	1	1	39	1	2	1	1	2.5	2	2	2
164	52691	Essakkiammal	22	1	1	38	1	2	1	1	2.5	2	2	2
165	52816	Nagalakshmi	31	1	2	38	1	2	1	1	2.4	2	2	2
166	45453	Fathima	24	2	1	37	1	2	1	2	1.6	2	2	2
167	51056	Selvi	23	1	1	36	1	2	1	1	2.7	2	2	2
168	53459	Manjula	21	1	1	37	1	2	1	1	2.3	2	2	2
169	49582	Selvi	24	1	2	36	1	2	1	2	2.5	2	2	2
170	53018	Natchiar	25	1	1	40	1	2	1	2	3.0	2	2	2
171	51008	Issaivani	19	1	1	38	1	2	1	1	2.8	2	2	2
172	45690	Sudha	21	1	1	37	1	2	1	1	2.7	2	2	2
173	46008	Mariammal	21	1	1	38	1	2	1	2	2.6	2	2	1

Sl. No	IP No:	Name	Age in years	Booked (1) / UnBooked (2)	Parity	Gestational Age in weeks	Status of Membranes Present 1 Absent 2	Mother culture Positive 1 Negative 2	Mode of Delivery Normal-1 Caesarean-2	Baby			Neonatal Outcome Morbidity Present 1 Absent 2	Post Partum Complications Present 1 Absent 2
										Sex Male-1 Female-2	Weight in Kg.	Culture Positive 1 Negative 2		
174	47018	Thangaeswari	28	1	2	40	1	2	2	1	2.5	2	2	2
175	47412	Sankara Avudaiamn	30	1	1	36	2	2	1	2	2.5	2	2	2
176	53648	Gomathy	25	1	2	37	2	2	1	1	3.0	2	2	2
177	52592	Vasantha	22	2	2	36	2	2	2	1	2.0	2	2	1
178	47598	Muppidathy	31	1	2	30	2	2	1	2	2.7	2	2	2
179	39102	Ponmani	25	2	1	28	2	2	1	1	2.0	2	2	2
180	45067	Mahalakshmi	23	1	2	35	2	2	1	1	1.5	2	2	2
181	46169	Gomathi	23	1	1	37	2	2	1	1	2.8	2	2	2
182	40741	Sudalaivadivoo	24	1	1	36	2	2	1	1	2.1	2	2	2
183	46747	Rajathi	20	1	1	30	2	2	1	2	2.5	2	2	2
184	35405	Alagumani	23	1	1	28	2	2	1	1	2.5	2	2	2
185	52581	Divya	19	1	1	30	2	2	1	1	2.2	2	2	2
186	52630	Jayanthi	21	1	1	29	2	2	1	1	2.7	2	2	2
187	52573	Gowri Thangam	23	2	2	28	2	2	1	2	2.3	2	2	2
188	52975	Mariammal	23	2	1	37	2	2	1	1	2.0	2	2	2
189	52950	Kalarani	24	1	1	36	2	1	1	2	2.5	2	2	2
190	52949	Poomani	20	1	1	36	1	2	1	1	2.0	2	2	1
191	44067	Nalini	22	1	2	28	2	2	1	1	2.2	2	2	2
192	41273	Anbukhani	26	2	1	35	2	1	1	2	2.0	2	2	2
193	52755	Mahalakshmi	24	1	1	36	2	2	1	1	2.1	2	2	2
194	37395	Mariammal	28	1	1	34	2	2	1	1	2.0	2	2	2
195	52932	Pushpharani	30	1	2	36	2	2	1	1	3.0	2	2	2
196	48648	Poomani	21	1	1	35	2	2	1	1	2.1	2	2	2
197	43863	Subbulakhsmi	29	2	2	36	2	2	1	1	2.0	2	2	2
198	52493	Essakkiammal	24	1	1	30	1	2	2	2	3.0	2	2	2
199	46130	Muthuselvi	25	1	1	36	2	2	1	1	2.5	2	2	2
200	45008	Kanagalakhsmi	26	1	1	28	1	2	1	1	2.6	2	2	2